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Vol. 5 FEBRUARY, 1936 No. 3

The Journal of Thoracic Surgery

Official Organ

The American Association for Thoracic Surgery

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Published Bi-Monthly by
The C. V. Mosby Company, 3523 Pine Blvd., St. Louis, U. S. A.

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The Journal of Thoracic Surgery

VOL. 5 FEBRUARY, 1936 No. 3

Original Communications

PLEUROPULMONARY COMPLICATIONS OF AMEBIASIS

AN ANALYSIS OF 153 COLLECTED AND 15 PERSONAL CASES*

ALTON OCHSNER, M.D., AND MICHAEL DEBAKEY, M.D.
NEW ORLEANS, LA.

UNTIL relatively recently amebiasis attracted little attention in non-tropical localities; although as early as 1902 Sir Leonard Rogers[1] demonstrated that complicating hepatic abscess, which previously had been designated "tropical abscess," occurred in other than tropical climates. The recent epidemic of amebiasis has more forcefully emphasized its ubiquitous distribution; although it is our opinion that the actual incidence of amebiasis in temperate climates probably has not increased in recent years. According to the authoritative opinion of Colonel Craig,[2] 10 per cent of the people of this country habor the Endameba histolytica, and frequently the diagnosis of amebiasis is overlooked because of the mildness of the clinical manifestations.

Amebiasis is of significance to the surgeon because of the relatively high incidence of hepatic and the less frequent incidence of pulmonary suppuration. The importance of pleuropulmonary complications of amebiasis cannot be sufficiently appreciated without some knowledge concerning the high incidence of hepatic amebic abscesses, because thoracic complications are generally the result of extension of the suppurative process in the liver through the diaphragm into the thorax. In a previous study on amebic hepatic suppuration we[3] collected 4,392 fatal cases of amebiasis, of which 37.9 per cent (1,664 cases) were complicated by hepatic suppuration. In our study there were 59 clinical cases of amebic abscess of the liver among 388 cases of amebic dysentery admitted to the Charity Hos-

*From the Departments of Surgery, Tulane University School of Medicine, Touro Infirmary, and Charity Hospital.

Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery, New York, June 3, 4, and 5, 1935.

pital in New Orleans during the six-year period 1928 to 1933, inclusive, an incidence of 15.2 per cent. Seven (13.5 per cent) of our 59 cases of amebic hepatic abscess were complicated by extension of the suppurative process to the thorax with involvement of the lung in 6 and the pleural cavity in 1. Pleuropulmonary amebic infections are second to hepatic lesions as complications of amebiasis. The present report is based on a study of 15 cases of pleuropulmonary complications of amebiasis admitted to the Charity Hospital in New Orleans from January 1, 1928, to April 1, 1935, and an analysis of 153 cases collected from the literature.

Historical.—According to Lemierre and Bernard,[4] Pringle in 1752, Portal in 1813, Larrey in 1829, Budd in 1845, and Cambay in 1847 observed cases in which hepatic abscesses, probably amebic, ruptured into the pleural cavity. Similar cases were observed by Duplay[5] in 1829 and by Behier[6] in 1836. Gaillard,[7] in 1856, reported a case of abscess of the liver with perforation into the pleura and the pericardium. Rouis,[8] in 1860, reported that in 162 autopsies on patients with amebic abscess of the liver 11 had perforated into the pleura. Other cases of historical interest are included among the collected cases analyzed in the present study (Hospel,[9] Rouis,[8] Foiret,[10] Bellot,[11] Gouran,[12] Morvan,[13] Debergues,[14] Gueschwind,[15] Simon,[16] Lafleur,[17] Ferrhon,[18] Bertrand and Fontan[19] and Osler.[20]

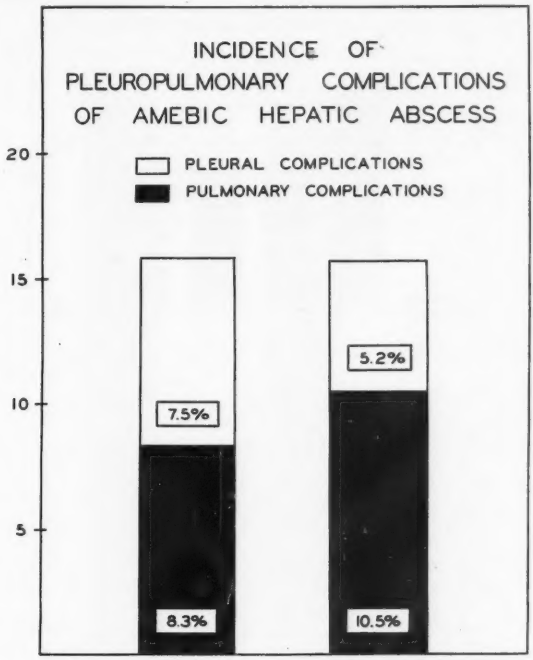
Incidence.—The frequency of pulmonary complications in amebiasis varies considerably according to different statistics. The incidence reported by different authors varies from 3 per cent in Strong's[21] series of

TABLE I

INCIDENCE OF PLEUROPULMONARY COMPLICATIONS IN REPORTED SERIES

	CASES OF LIVER ABSCESS	LUNG COMPLICATIONS	PLEURAL COMPLICATIONS
Clark[23]	95	5	
Craig[24]	22	1	7
Waring[25]	300	15	28
Dutroulan[26]	66	7	10
Roux[27]	162	14	17
Hospel[9]	25		2
Cambay[28]	10		2
Futcher[29]	27	9	3
Sambuc[30]	102	7	4
Murray and Koh[31]	23	4	
Strong[21]	100	1	2
Thompson[32]	27	7	4
Rendu[33]	563		31
Harris[34]	95		3
Talbot[35]	15	1	
Vergoz-Hermemjat-Gerin[36]	80		28
Thierfelder[22]	170	74	26
Cyr[37]	563	59	31
Manson-Bahr[38]	45	5	
	2,490	209 (8.3%)	198 (7.5%)
Authors[3]	95	10 (10.2%)	5 (5.2%)

100 fatal cases of amebiasis in which the lung was involved once and the pleura twice, to 58.8 per cent of 170 cases reported by Thierfelder.[22] In a series of 2,490 reported amebic hepatic abscesses, pleural complications occurred in 198 (7.5 per cent) and pulmonary complications in 209 (8.3 per cent) (Cases 3, 9, 21, 22, 28-38) (Table I). Of 95 consecutive cases with amebic hepatic abscess admitted to the Charity Hospital and the Touro Infirmary in New Orleans from January 1, 1928, to April 1, 1935, 15 (15.7 per cent) had pleuropulmonary complications. In 7 (7.3 per cent) the hepatic abscess perforated into the lung, in 5 (5.2 per cent)



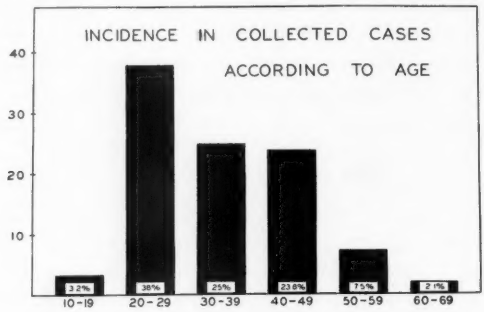
Graph 1.—Graph showing incidence of pleuropulmonary complications in collected series and in authors' series.

into the pleura, and in 3 (3.1 per cent) there was a bronchopleural fistula. The total incidence of pleuropulmonary complications in our series (15.7 per cent) is practically the same as that obtained in the collected series (15.8 per cent) (Graph 1).

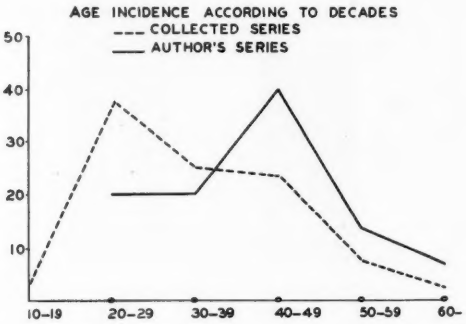
Pleuropulmonary amebic infections occur most frequently in the third and fourth decades of life. In the collected series of cases there were 92 in which the age was stated. Of this group, 3 (3.2 per cent) were in the second, 35 (38 per cent) in the third, 23 (25 per cent) in the fourth, 22 (23.8 per cent) in the fifth, 7 (7.5 per cent) in the sixth, and 2 (2.1 per cent) in the seventh decades (Graph 2). The youngest was four-

teen years old, the oldest sixty-two years, and the average age was thirty-four and five-tenths years. In our series the youngest was twenty-one, the oldest sixty-three, and the average age was forty and six-tenths years. Three (20 per cent) were in the third decade, 3 (20 per cent) in the fourth, 6 (40 per cent) in the fifth, 2 (13.3 per cent) in the sixth, and 1 (6.6 per cent) in the seventh decades. The majority of our patients were approximately two decades older than in the collected cases (Graph 3), a fact which we are unable to explain.

The sex incidence is similar, as would be expected, to that of amebic abscess of the liver, i.e., a preponderant occurrence in males. In the



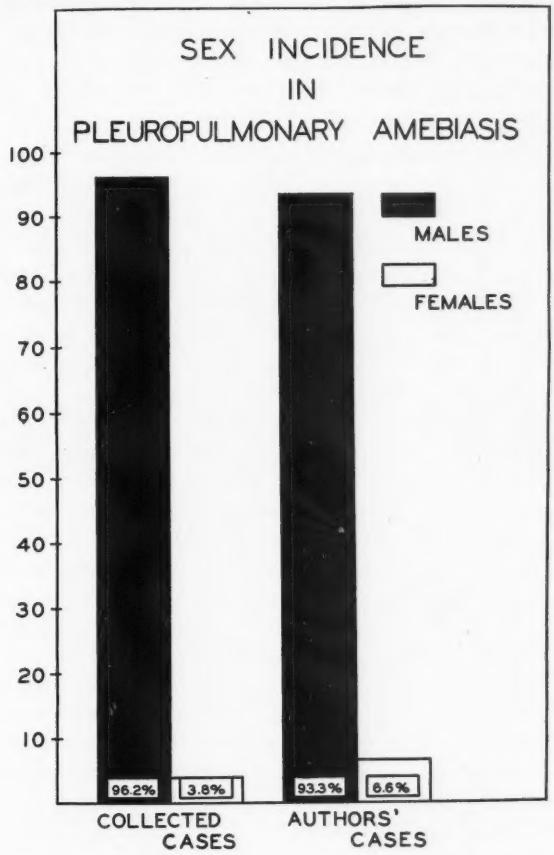
Graph 2.—Graph showing age incidence in collected series.



Graph 3.—Graph showing comparison of age incidence in collected series and in authors' series.

collected cases the sex was stated in 120, of which 115 (96.2 per cent) were males and 5 (3.8 per cent) females. In our series there were 14 males and 1 female (Graph 4). This considerably higher incidence in males is explained by the apparently greater susceptibility of males to amebic abscess of the liver. In the collected cases there were 4 in which it was stated the patient was colored and 14 were white. Presumably the other 135 were white. In our series of 15 cases there were 9 white and 6 colored patients.

Etiology and Pathogenesis.—Pleuropulmonary amebic involvement is probably always secondary to amebiasis elsewhere and is generally the result of extension of the process from an amebic infection of the liver. Most, if not all, amebic infections begin in the large bowel, from which area the amebas may be carried probably through the blood stream to other parts of the body. As the intestinal tract is drained by the portal



Graph 4.—Graph showing sex incidence in collected series and in authors' series.

vein, involvement of the liver occurs relatively frequently. In most instances the amebic infection of the pleura and the lungs is the result of direct extension from an amebic hepatic abscess, perforation occurring through the diaphragm into the lung, bronchus, or pleura. Rarely transphrenic migration of the amebas from a subphrenic amebic process to the thorax may occur without gross perforation. This according to Loison[39] is the result of amebas being carried in the diaphragmatic

lymphatics. Relatively rarely involvement of the pulmonary parenchyma may occur as a result of hematogenous infection, the amebas gaining entrance to the lung either by the blood stream or through the lymphatics and blood stream. In the former, amebas may be carried through the portal vein, hepatic capillaries, and hepatic veins to the vena cava or through the anastomotic collaterals of the hemorrhoidal veins into the vena cava (Petzetakis[40]). In the latter the amebas may be carried from the colon through the thoracic duct to the left subclavian vein and hence to the lungs. Strong,[21] on the other hand, is of the opinion that the pleuropulmonary complications are never metastatic, but always occur by direct extension from a liver abscess.

Rarely, if ever, primary amebic infection of the lung occurs. Panayatalou and Netter[41] described a case which they considered primary amebic bronchitis. Petzetakis[42] described 2 cases of amebic bronchitis in which amebas were found in the sputum, one of which was not associated with any bowel disturbance. A similar case was reported by Haberfeld,[43] who believes that although there is no history of amebic infection of the bowel, one is not justified in assuming such never was present. In many of the reported cases a liver abscess and pulmonary abscess occurred concomitantly without any apparent connection between the two. The majority of amebic abscesses of the liver occur on the convex surface and in the posterior portion of the right lobe; and as the abscess enlarges, its periphery approaches the diaphragm, gradually incorporating it in the wall of the abscess, facilitating the perforation of the hepatic suppurative process into the thoracic cavity. In most instances there is an accompanying pleurisy with adhesions which obliterate the pleural cavity, so that rupture occurs into the base of the right lung. In those instances, however, in which extension of the hepatic process is more rapid and in which there is no antecedent pleurisy, the abscess ruptures into the pleural cavity. Many times rupture of an hepatic abscess into the lung produces relatively little inflammatory pulmonary reaction, the contents of the hepatic abscess being evacuated through the communication between the liver abscess and a bronchus. In such instances the clinical manifestations rapidly subside, especially under adequate therapy.

Pathology.—There are a number of types of pleuropulmonary amebic infections, and in our study of the collected cases we have divided the lesions into five different groups, depending upon the type of the pleuropulmonary involvement: (1) hematogenous pulmonary abscess without liver involvement; (2) hematogenous pulmonary abscess and independent liver abscess; (3) pulmonary abscess extending from liver abscess; (4) bronchohepatic fistula with little pulmonary involvement; (5) empyema extending from liver abscess (Fig. 1). In our collected series of 153 cases there were 22 in which the pulmonary amebic infection

occurred without liver involvement (Fig. 1). In one of these reported by Panayatalou and Netter[41] (Case 115), the amebic infection of the lung was supposedly primary. In the other 21 the amebic infection was embolic and secondary to a process in the colon. Kofoid, Boyers, and Swezy[44] believe that hematogenous transportation of amebas occurs more frequently than is generally supposed. In the investigations in fatal cases they found amebas in practically every tissue of the body examined. Strong,[21] on the other hand, contends that amebic infections are never embolic and believes in those cases in which a communication between the hepatic abscess and the thoracic lesion cannot be demonstrated that

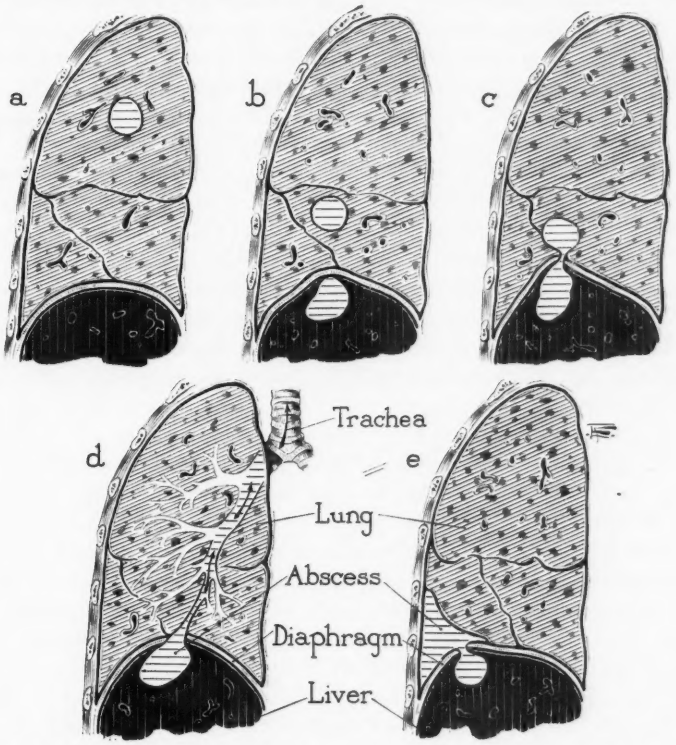


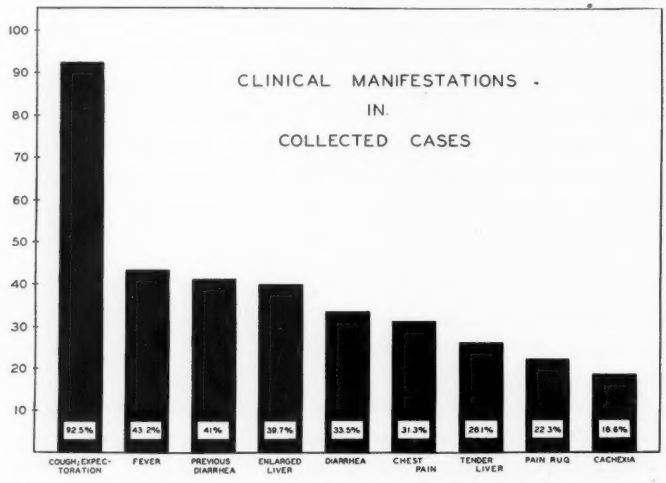
Fig. 1.—Diagrammatic representation of various types of pleuropulmonary amebic infections. *a*, Hematogenous pulmonary abscess without liver involvement; 14.3 per cent of the collected series and none of our cases were of this type. *b*, Hematogenous pulmonary abscess and independent liver abscess. As illustrated in the drawing the pulmonary process is separated from the hepatic process by normal lung; 10.4 per cent of the collected series and none of our cases were of this type. *c*, Pulmonary abscess extending from liver abscess. In contrast to Group 2, intervening portion of lung between pulmonary and hepatic process is involved; 37.2 per cent of the collected series and 46.6 per cent of our cases were of this type. *d*, Bronchohepatic fistula with little pulmonary involvement. In this type, communication between hepatic abscess and bronchus is more direct and large, permitting evacuation of hepatic abscess contents with little involvement of pulmonary parenchyma; 19.6 per cent of the collected series and 20 per cent of our cases were of this type. *e*, Empyema extending from liver abscess. Because of absence of pleural adhesions, perforation of hepatic process occurs into pleural cavity; 17.6 per cent of the collected and 33.3 per cent of our cases were of this type.

the perforation has healed. In the collected series there were 16 cases in which a hematogenous pulmonary abscess and a liver abscess occurred concomitantly without any apparent communication between the two (Fig. 1). There were 5 of the group of 16 cases in which a perforation of the liver abscess into the pleura or lung occurred, although the original lung abscess in the 16 cases was supposedly hematogenous. In 4 of these (Cases 77, 150, 151, 152) the liver abscess ruptured into the pleural cavity; whereas in 1 (Case 57), the liver abscess ruptured into the lung. In by far the greater majority of cases the amebic infection of the lung occurs in the right lower lobe. This is probably due to the fact that in most instances the infection gains entrance to the lung by direct extension from the liver. In our previous study[3] on amebic hepatitis we found the right lobe of the liver involved in 92.9 per cent. Adhesions are usually present between the inferior surface of the right lower lobe of the lung and the diaphragm and between the superior surface of the liver and the diaphragm so that perforation of liver abscess is usually directly into the lung or bronchus without passing through the uninvolved pleural cavity. In cases with no limiting adhesions, perforation of the liver abscess into the pleural cavity occurs. In the collected series of 153 cases there were 58 in which a liver abscess had extended into the lung, producing amebic pneumonitis and abscess (Fig. 1). In each series the pulmonary abscess was located in the lower lobe. In our series 7 of the 15 were of this type. Occasionally perforation of the liver abscess into the lung occurs in the region of a large bronchus with the development of a bronchohepatic fistula which permits adequate evacuation of the liver abscess and produces little reaction in the pulmonary parenchyma (Fig. 1). In the collected series of 153 cases there were 30 patients with bronchohepatic fistulas and with little or no pulmonary involvement. In our series there were 3 of this type (Cases A 3, A 11, A 12). These cases must be distinguished from the communicating hepatic and pulmonary abscesses in which there is also a communication with a bronchus. In the collected series of cases there were 27 in which a liver abscess was complicated by empyema (Fig. 1). In our series of 15 cases, 5 were of this type. In those cases in which a localized abscess in the inferior portion of the pleural cavity communicates with an abscess below the diaphragm, Pruvost[45] describes the abscess as a "shirt stud" abscess. Strong[21] has described the amebic liver abscess as being filled with a grayish yellow or yellowish red fluid material. He differentiates between amebic pulmonary abscess and liver abscess in that the walls of the former are less even than those of the latter. In some areas the walls are smooth and are composed of connective tissue. He describes three zones in the walls of the older abscesses, the first, a necrotic one containing fragmented nuclei, degenerated cells, and amebas. The second layer is composed of connective tissue fibers, epithelial cells, elastic fibers, and groups of alveoli with occasional amebas.

The third consists of a layer of small round cell infiltration in which fibrin and some proliferation of connective tissue fibers are visible between the alveoli. The walls of the bronchi in the region of the abscesses are infiltrated with numerous round cells, and the bronchi usually contain purulent or serous fluid. The contents of the abscess may vary according to its type. In the hematogenous amebic abscess the contents are purulent and are in no manner characteristic; whereas in those cases in which a communication between the liver abscess and the pulmonary or pleural lesion exists, the contents are usually chocolate sauce pus.

Clinical Manifestations.—Clinical manifestations of amebic infections of the lung and pleura vary according to the mode of the infection. In those cases in which the infection extends from a liver abscess, the first manifestation is usually a severe pain in the lower portion of the chest on the right side. This is undoubtedly due to the involvement of the diaphragmatic pleura by the inflammatory process. Bernard[46] has emphasized the importance of shooting pains at the right base accompanied by slight pyrexia. Pruvost[45] describes the pain as violent and as being aggravated by respiration. The pain is frequently referred to the right shoulder, a symptom which was especially emphasized by Thompson.[32] A distressing, unproductive cough in the beginning, probably due to pleural involvement, is a prominent symptom. Expectoration of large quantities of purulent material and particularly chocolate sauce pus occurs in cases with communication between the abscess and a bronchus. Dyspnea may occur, especially in cases in which there is a communication with a bronchus and in which there is considerable encroachment upon a large portion of the lung field. In the 153 collected cases a statement was made concerning symptoms in 134. The chief complaint was cough and expectoration in 87 (64.9 per cent), fever in 24 (17.9 per cent), pain in the chest in 21 (15.6 per cent), pain in the right upper quadrant in 15 (11.1 per cent), diarrhea in 13 (9.5 per cent), pain in the right shoulder in 4 (2.9 per cent), loss in weight in 3 (2.2 per cent), dyspnea in 2 (1.4 per cent), malaise in 2 (1.4 per cent), swelling of the right chest in 1 (0.7 per cent), and weakness in 1 (0.7 per cent). In our cases the chief complaints were as follows: cough and expectoration in 6 (40 per cent), pain in the chest in 6 (40 per cent), pain in the right upper quadrant in 4 (26.6 per cent), fever in 1 (6.6 per cent), and pain in the right shoulder in 1 (6.6 per cent). Aside from the chief complaint there were other symptoms described in 134 of the 153 collected cases. One hundred and twenty-four (92.5 per cent) of these complained of cough and expectoration, 58 (43.2 per cent) of fever, 55 (41 per cent) of a previous diarrhea (Graph 5). In 1 case a previous diarrhea was denied. Forty-six patients (33.5 per cent) had diarrhea at the time of admission to the hospital. Forty-two (31.3 per cent) had chest pain. Thirty (22.3

per cent) had pain in the right upper quadrant. Twenty-five (18.6 per cent) had cachexia. Twenty-one (15.6 per cent) had dyspnea. Seventeen (12.6 per cent) had pain in the right shoulder. In our 15 patients all (100 per cent) had fever. Fourteen (93.3 per cent) had cough and expectoration. Five (33.3 per cent) had pain in the chest. Four (26.6 per cent) had pain in the upper right quadrant. Three (20 per cent) had pain referred to the right shoulder. Three (20 per cent) had dyspnea. Only 2 (13.3 per cent) had had previous diarrhea, and only 1 (6.6 per cent) had diarrhea during the present illness. Two (13.3 per cent) were emaciated, and 1 (6.6 per cent) had chills. Profuse expectoration of chocolate sauce pus is pathognomonic in amebic infections of the lung and indicates rupture of amebic hepatic abscess into a bronchus. In 74 instances of the 153 collected cases it was stated



Graph 5.—Graph showing incidence of clinical manifestations in collected cases.

definitively that chocolate sauce pus was present in the sputum. In the majority of other instances no statement was made concerning the character of the expectorated material. In our series of cases chocolate sauce pus expectoration was present in 14 cases, an incidence of 100 per cent, as there were only 14 with bronchial communication. The fifteenth case in our series was one with a perforation of the hepatic abscess into the pleural cavity alone.

The chest findings are usually those of consolidation and cavitation. As a matter of fact, in many instances a diagnosis of pulmonary tuberculosis is made erroneously. Dullness over the affected portion of the lung is the most frequent pulmonary sign. Statements regarding physical signs were made in 134 of the 153 collected cases. In 33 of the collected series and in 9 of our 15 cases there was thoracic dullness.

Râles were found in 17 of the reported cases and in 9 of our cases. Diminished breath sounds were found in 9 of the collected cases and in 3 of our cases. In those relatively rare cases in which the liver abscess first perforates into the pleural cavity with the development of an empyema, the clinical manifestations are those of pleural effusion, i.e., dullness, diminished breath sounds, and limitation of motion of the involved hemithorax. In addition to the thoracic manifestations the abdominal manifestations of amebic infection are important. In the majority of instances the liver is enlarged and tender. The liver was enlarged in 53 (39.7 per cent) and tender in 35 (26.1 per cent) of the collected cases. The same signs were found in 6 (40 per cent) of our 15 cases. The absence of liver enlargement was explicitly stated in 5 of the collected cases. The finding of concomitant involvement of the liver and lung is suggestive of amebic pulmonary infection. In practically all

Graph 6.—Graph showing incidence of positive findings of amebas in sputum and pus of collected series. The percentages are based on the number in which a statement was made concerning this examination.

cases there is a moderate pyrexia. Characteristically, however, as in the amebic infections elsewhere in the body, the elevation of the temperature is not high. In our series of cases the highest temperature on admission was 103° F. In the majority, however, it ranged between 100° and 101° F. Hyperpyrexia and chills in cases of amebic infections of the pleura and lungs probably indicate secondary infection and are not caused by the amebic infection per se.

Laboratory Findings.—As in amebiasis elsewhere in the body, a moderate leucocytosis is usually encountered in amebic infections of the pleura and lungs. In our previous publications[3, 47] we emphasized that in amebic hepatic suppuration there is a moderate leucocytosis without a concomitant increase in the polymorphonuclear leucocytes. In the collected cases of pleuropulmonary amebic infections the highest leucocyte count observed was 52,000, the lowest 9,700, the average 18,860.

The highest polymorphonuclear count was 91 per cent, the lowest 53 per cent, and the average 72.8 per cent. In our series of cases the highest leucocyte count was 51,250, the lowest 7,500, the average 17,000. The corresponding polymorphonuclear percentages were 92 per cent, 70 per cent, and 79 per cent, respectively. Obviously the leucocyte count and polymorphonuclear percentage are not so high in amebic infections of the lung as in corresponding pyogenic infections, especially pneumococcic. In those cases of amebic infections of the lung which are accompanied by high leucocyte and polymorphonuclear counts a secondary infection probably has supervened. In recent pulmonary amebic infections, amebas are generally present in the sputum and can be demonstrated by microscopic examination. Petzetakis[48] emphasizes the importance of immediate examination of the sputum. A moist, warm preparation is best for the microscopic study. In the collected cases amebas were found in the sputum in 19, and in 5 cases it was explicitly stated that amebas were not present (Graph 6). In 7 of our cases the sputum was examined for amebas, and it was found positive in 2 (28.5 per cent). The stools may or may not contain amebas or cysts. The reason there are not more positive stool findings is that frequently the colonic amebic infection has subsided by the time the pulmonary symptoms develop. In the collected cases the stools were examined in 8, and amebas were found in 2. According to Futcher,[29] the sputum in these cases contains pus cells, elastic tissue, hematoidin crystals, and occasionally Charcot-Leyden crystals. Bernard[46] believes that a diagnostic point in regard to the sputum is the small number of organisms which are in the sputum early in the condition, although later, as a result of secondary infection, many organisms may be present.

Roentgenologic Examination.—X-ray examinations in amebic infections of the lung and pleura are quite characteristic, especially those in which perforation of a liver abscess has occurred into the lung. In the latter instance, prior to the perforation into the thorax, liver abscess produces a localized bulging of the diaphragm (Figs. 2 and 3). In cases, however, in which the intrahepatic abscess has ruptured into the subphrenic space with the development of a hepatic subphrenic abscess there is elevation of the diaphragm, especially in the cardiophrenic portion in the anterior posterior roentgenogram and an obliteration of the anterior costophrenic angle in the lateral roentgenogram (Granger,[49] Ochsner and DeBakey[47]). In those cases in which perforation into the lung has occurred a triangular shadow with the base toward the liver can usually be visualized in the roentgenogram (Figs. 4 and 5). This is particularly true in the lateral roentgenograms, although it is also seen in the anteroposterior roentgenogram. Our Case A 12 demonstrates these findings especially well. Thoracic roentgenograms taken while the patient was ambulatory showed typical localized bulging of the dia-

Fig. 2. Fig. 3.

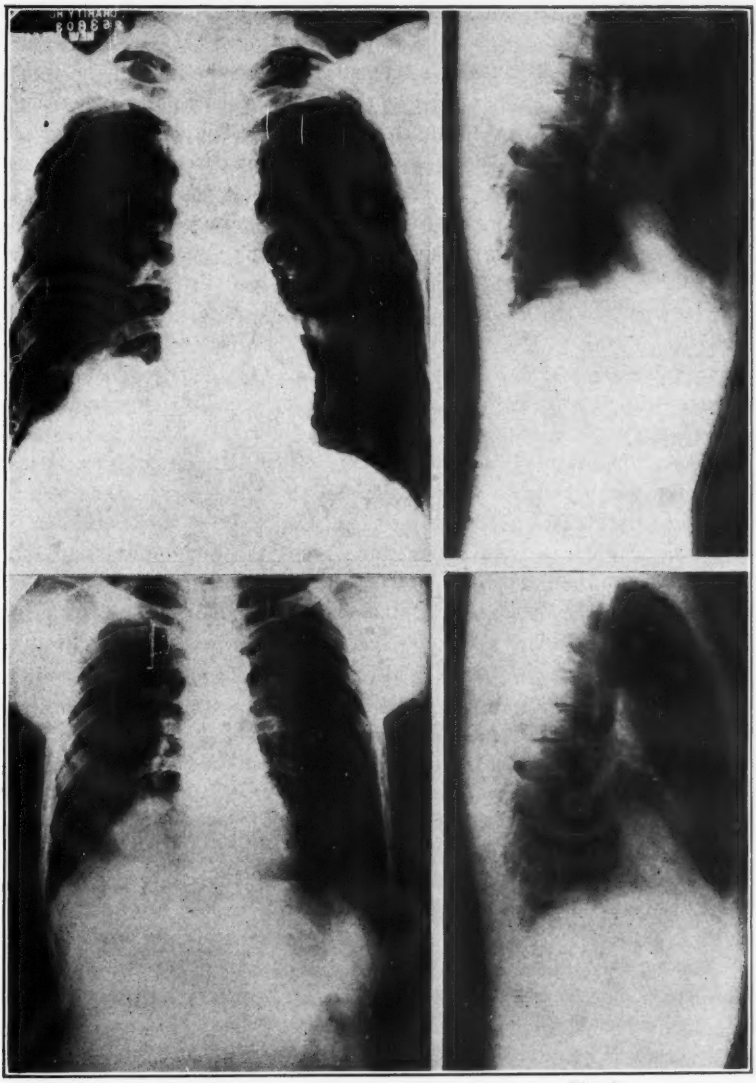


Fig. 4. Fig. 5.

Fig. 2.—Anteroposterior roentgenogram of Case A 12 with amebic hepatitis, showing localized upward bulging of diaphragm in its medial portion.

Fig. 3.—Lateral roentgenogram of same patient as in Fig. 2, showing triangular shadow in lower portion of thorax with base below and apex pointing upward and posteriorly.

Fig. 4.—Anteroposterior roentgenogram of Case A 12 (shown in Fig. 2) taken several hours after perforation of amebic hepatic abscess into the lung and bronchus. In contrast to the shadow in Fig. 2, the edges of which are sharply defined, the shadow at the right base in this roentgenogram is less distinct, particularly in the upper portion.

Fig. 5.—Lateral roentgenogram of Case A 12 (shown in Fig. 3) taken several hours after perforation of amebic hepatic abscess. The shadow at the right base has extended upward and in contrast to the shadow in Fig. 3 the edges are less distinct, especially in its uppermost portion.

phragm into the lower lung field (Figs. 2 and 3). Hospitalization was advised, but was deferred by the patient for two weeks. On the day of admission the patient experienced a severe pain at the base of the right thorax and began to expectorate large quantities of chocolate sauce material. Roentgenograms taken approximately three hours after the onset of these symptoms showed extension, especially upward, of the previously demonstrated amebic hepatic abscess and irregularity of the apex of the shadow which on previous examination was sharply defined (Figs. 4 and 5). In cases with hematogenous lung abscess the shadow may be located in any part of the lung, and usually there is a clear zone between the shadow produced by the abscess and that produced by the liver. Flynn and Warren[50] believe the following are cardinal roentgenographic signs of bronchohepatic fistulas: (1) high fixed right diaphragm; (2) abscess mass near the diaphragm and involving the liver; (3) column of fluid extending from this to the trachea; and (4) expectoration of bile-stained sputum. In the collected series of cases relatively few x-ray pictures were made. In 19 of the group there was a shadow at the right base; in 10 the right diaphragm was elevated; in 6 there was evidence of abscess in portions of the lung other than the base, and in 1 there was evidence of fistula between the liver and the lung. In our series of 15 cases a shadow at the right base was found in 12, there was elevation of the diaphragm in 11, abscess of the lung in 3, and abscess with fistula in 2. In these last 2 cases the fistula was demonstrated by means of lipiodol filling.

Diagnosis.—The diagnosis of amebic infections of the pleura and lung are usually not difficult if the condition is only kept in mind. Generally, there is a history of previous diarrhea, moderate elevation of the temperature, and enlargement and tenderness of the liver. When these symptoms and signs are associated with pulmonary manifestations, one should suspect an amebic infection of the lung. If, in addition to this, there are elevation and fixation of the diaphragm and a basal shadow on the right side together with expectoration of large quantities of chocolate sauce pus, one is justified in making a positive diagnosis. We have found, as suggested by Pellé and Baron[51] and Lemierre and Bernard,[4, 46] that the well-defined edge of the roentgenographic shadow is of diagnostic value (Figs. 4 and 5). Cannavò and Cola[52] emphasize the advantage of using contrast substances introduced into the bronchial tree perorally to visualize the communications from the liver, pulmonary abscesses, and the bronchi. In 2 of our cases the fistulous communication between the hepatic abscess and the bronchi was demonstrated by lipiodol filling of the tracheobronchial tree. Cannavò and Cola[52] also advocate the use of pneumoperitoneum and pneumothorax as diagnostic procedures. The finding of amebas in the sputum together with relatively few microorganisms is conclusive proof of the correct diagnosis.

The demonstration of a clear zone between the pulmonary shadow and that produced by the liver is indicative of a hematogenous abscess. In those cases in which a communication between the amebic process and a bronchus has not been established, as in empyema resulting from perforation of an hepatic abscess, aspiration of the fluid is a valuable diagnostic procedure. The finding of characteristic sterile chocolate sauce pus, especially if it contains amebas, is pathognomonic. Amebas, however, are found infrequently in the aspirated material. In the collected series they were found in 11 of the cases (Graph 6). In our 15 cases the aspirated material was examined for amebas in 5, in 4 (80 per cent) of which negative results were obtained. In only 1 (Case A 4) were amebas in the aspirated material.

Amebic infections of the lung are likely to be confused with tuberculosis, primarily because of the chronicity of the condition and because of the profuse expectoration of bloody sputum. The physical findings of consolidation and cavitation support this diagnosis. In tuberculosis, however, the greatest amount of involvement is usually at the apex; whereas in amebiasis the lesion is generally at the base and associated with hepatic involvement. Tuberculosis can easily be eliminated as a diagnostic possibility by the absence of tubercle bacilli in the sputum. Some cases, because of the acute onset, are confused with pleurisy, and pneumonia. It is exceptional, however, that the high leucocyte count seen in the pyogenic pneumonias occurs in cases of amebic pneumonitis; and, when such does occur, it is usually indicative of secondary infection with pyogenic bacteria. Then, too, as a rule, the amount of material expectorated in cases of amebic infections of the lung is considerably greater than that expectorated in pyogenic infections. The temperature reaction in amebic infections is considerably lower than that in the usual pyogenic infections. Characteristically the fever in amebic pneumonitis is only moderately elevated, and the patient, although cachectic, usually is not so ill as the patient with a pneumonic process. The presence of characteristic chocolate sauce pus in the sputum is diagnostic of amebic hepatic suppuration communicating with a bronchus and is of value in distinguishing such lesions from other pulmonary processes. Rarely the sputum is bile stained. In suspected pleuropulmonary complications of amebic hepatic abscess, aspiration of the hepatic abscess is justified. This should be done, as emphasized in our previous communications,[3, 47] in such a way that an uninvolved portion of a serous cavity is not traversed by the aspirating needle in order to prevent contamination of the respective cavity.

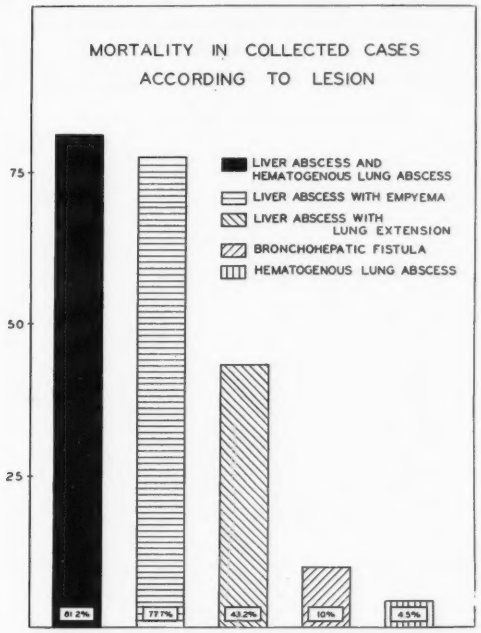
Prognosis.—The prognosis in amebic infections of the pleura and lung depends upon a number of different factors. Hussenet[53] and Valence[54] state that the prognosis in amebic pulmonary suppuration is dependent upon the presence or absence of bile in the sputum. When bile is present,

TABLE II

RESULTS IN COLLECTED SERIES

TYPE OF THERAPY	HEMATOGENOUS PULMONARY ABSCESS WITHOUT LIVER INVOLVEMENT		HEMATOGENOUS PULMONARY ABSCESS AND INDEPENDENT LIVER ABSCESS		PULMONARY ABSCESS EXTENDING FROM LIVER ABSCESS		BRONCHOHEPATIC FISTULA WITH LITTLE PULMONARY INVOLVEMENT		EMPYEMA EXTENDING FROM LIVER ABSCESS		TOTAL	
	RECOVERED	DIED	RECOVERED	DIED	RECOVERED	DIED	RECOVERED	DIED	RECOVERED	DIED	RECOVERED	DIED
Operation	63			43, 45, 57, 58, 59, 77, 106	29, 30, 40, 41, 42, 52, 53, 54, 61, 62, 66, 67, 81, 83, 92, 95, 99	46, 47, 49, 55, 56, 64, 65, 69, 82, 86, 96, 98, 100	19, 28, 44, 68, 70, 73, 75, 128	36, 37	22, 76, 97	14, 17, 51, 74, 89	29	27
Operation and emetine	117		150	151, 152	87, 127, 137, 146		85, 108, 132		107		10	2
Emetine	109, 110, 113, 114, 115, 116, 118, 119, 122, 129, 133, 135, 136, 138, 141, 143, 144, 145, 153		111, 112	123	84, 125, 134, 139, 140, 142	130	79, 80, 126, 131, 147, 148, 149		105		35	2
No statement made or insufficient data		48		60, 88, 121	20, 33, 39, 94, 120	6, 9, 12, 18, 21, 23, 24, 31, 34, 50, 93	16, 25, 26, 27, 32, 35, 38, 71, 72	15	90	1, 2, 3, 4, 5, 7, 8, 10, 11, 13, 91, 101, 102, 103, 104, 124	15	32
Postural drainage					78						1	
Total	21	1	3	13	33	25	27	3	6	21	90	63
	22		16		58		30		27		153	

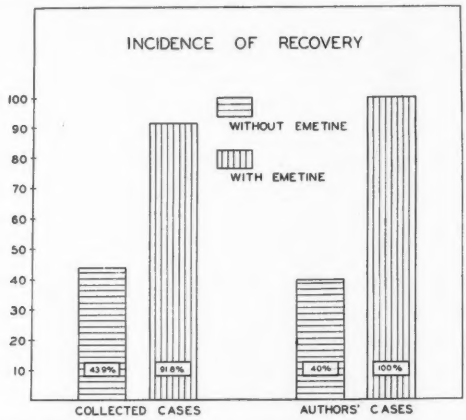
the prognosis is much worse than when it is absent. The type of pleuropulmonary involvement is important as regards prognosis, the prognosis being gravest in those cases in which an hepatic abscess ruptures into the pleural cavity and best in those cases in which there is a direct communication between the hepatic abscess and a large bronchus with a minimal amount of pulmonary reaction. According to Thompson[32] the chances of spontaneous cure are fairly good in those cases with a communication between the liver abscess and the bronchus without involvement of the pleura; whereas, if the pleural cavity is involved, there is little chance of spontaneous cure. The prognosis is dependent not only



Graph 7.—Graph showing mortality in collected cases in pleuropulmonary amebiasis according to type of lesion.

upon the type of the pleuropulmonary involvement but also upon the type of therapy. According to Miginiac,[55] one must differentiate between acute and chronic amebic pulmonary abscesses. In the former there is usually a large communication between the hepatic abscess and the bronchus, permitting rapid evacuation of the hepatic abscess contents; whereas in the latter the communication is small, not infrequently there are multiple abscesses, and as a result of the incomplete evacuation, hyperpyrexia, cachexia, anemia, and weakness persist. The prognosis in this type of case is bad. The importance of adequate drainage through the bronchus in cases of pleuropulmonary amebiasis is shown by the results

obtained in the collected cases. Of the 30 patients with liver abscess and bronchohepatic fistula with little or no involvement of the lung parenchyma, 27 recovered (90 per cent) (Table II, Graph 7). In contrast to the high incidence of recoveries in this group of cases is the low incidence of recoveries in those cases in which a liver abscess was complicated by an empyema. In the collected series there were 27 cases with only 6 recoveries (22.2 per cent) (Table II, Graph 7). The importance of the use of amebicides as regards the prognosis in amebic infections of the pleura and lung is illustrated by the results obtained in the collected cases; 91.9 per cent of the patients receiving emetine recovered as contrasted with 43.9 per cent recovery in those patients not receiving emetine. In our cases similar results were obtained in that there was a 40 per cent recovery in the patients not receiving emetine and 100 per cent recovery in those cases in which emetine was used (Graph 8). The effect

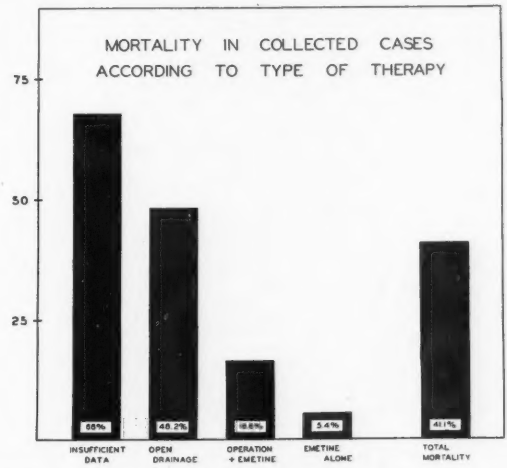


Graph 8.—Graph showing incidence of recoveries in collected series and in authors' series in those patients treated with and without emetine. The importance of emetine in the treatment is graphically illustrated.

of emetine administration on the prognosis is further illustrated by the results in the collected cases. In the cases treated by open drainage without amebicides the mortality rate was 48.2 per cent; whereas in those cases treated by open drainage and emetine the mortality rate dropped to 15.3 per cent, and only 5.5 per cent of the patients treated by emetine alone died (Graph 9). Theoretically the prognosis in hematogenous pulmonary abscess should be much worse than in those cases in which there is direct extension into the lung from the liver, because in the former the amebas are circulating in the blood stream. In the collected series of hematogenous lung abscess occurring concomitantly with liver suppuration there were 16 cases with 13 deaths, a mortality rate of 81.2 per cent. In the group of cases, however, in which a hematogenous abscess of the lung occurred without any involvement of the liver, there were 22

cases with 1 death, a mortality rate of 4.5 per cent. The great discrepancy in the results obtained in these two groups, both with amebic blood stream infections, is due to two factors: (1) patients with hematogenous lung abscess and concomitant liver abscess have two suppurative processes, either one of which is a burden for the organism to carry; (2) the type of therapy employed in each. We believe the latter factor to be the more important of the two. A large number in the group with the hematogenous lung abscess alone received emetine; whereas relatively few of those with concomitant liver abscess received emetine. In the former group of 22 cases all but 2 patients received emetine, and the only fatal case did not. In the latter group of 16 cases only 6 received emetine.

Treatment.—Before the suggestion of Sir Leonard Rogers,[56] in 1912, that emetine be employed in the treatment of amebic dysentery and



Graph 9.—Graph showing mortality in the collected series according to type of therapy.

amebic hepatitis the results obtained in the treatment of these conditions were unsatisfactory. Whereas at the present time there are many amebicides which are more efficacious and much safer in the treatment of amebic lesions in the bowel; emetine still remains the sheet anchor in the treatment of amebic infections of the liver and lung. As mentioned in our previous publication,[3] Leake[57] is of the opinion that although other amebicides (acetarsone, carbasone, trepasol, chinophon, and vioform) are safer and more efficient in the treatment of intestinal amebiasis, they should not be used in amebic hepatic infections, because they themselves are toxic to the liver. After the treatment with emetine and the subsidence of hepatic and pleuropulmonary manifestations, however, the underlying intestinal amebiasis is most safely and efficiently treated by one of the above mentioned amebicides. Manson-Bahr[58] has emphasized

that emetine is more efficacious in the treatment of pleuropulmonary amebiasis than in the intestinal lesion. The value of emetine in the treat-

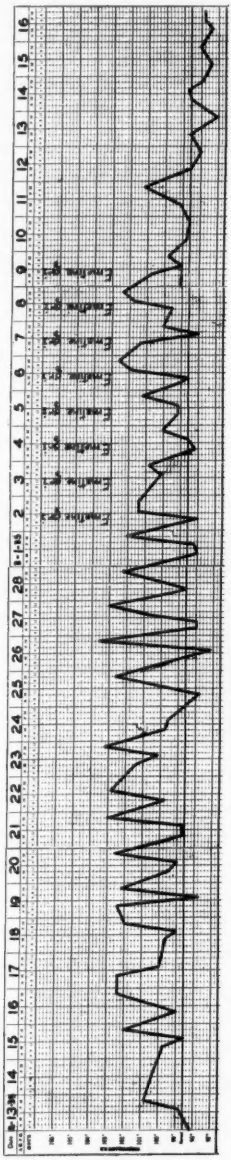


Fig. 6.—Temperature chart of Case A 12, showing septic type of fever and definitive response to emetine therapy.

ment of pleuropulmonary amebiasis is illustrated by the results obtained in the collected cases and in our cases (Figs. 6 and 7). In the former group there was a recovery incidence of 43.9 per cent in patients not re-

ceiving emetine as compared with 91.8 per cent in those who received emetine. In the latter group, the authors' cases, the recovery incidence in the group not receiving emetine was 40 per cent as compared with 100 per cent in those patients who received emetine (Graph 8). Similarly in the collected series the effect of the administration of emetine is illustrated by the comparison of results obtained in those cases in which

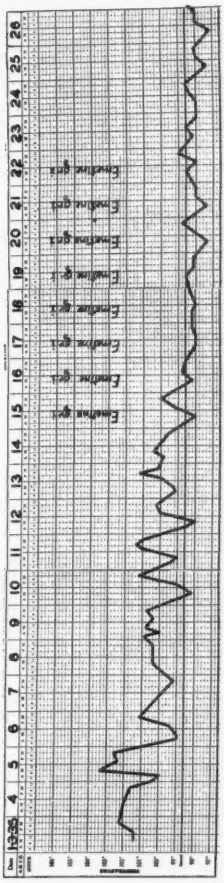
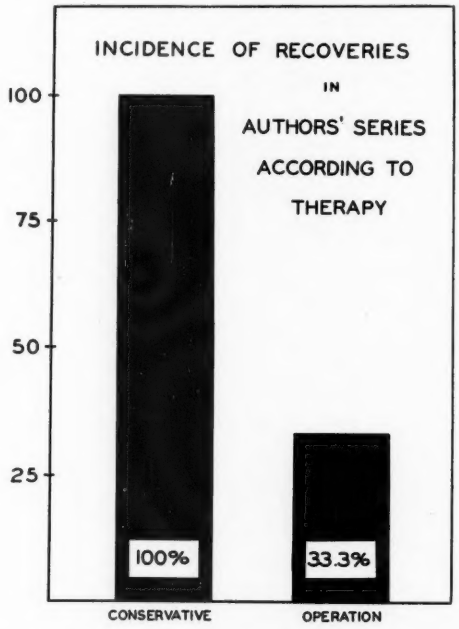


Fig. 7.—Temperature chart of Case A 13, showing moderate irregular pyrexia and dramatic response to emetine therapy.

open operation and drainage of the abscess was done with those in which the abscess was drained together with administration of emetine. In the former group the mortality rate was 48.2 per cent, whereas in the latter group it was 15.3 per cent, and in a third group of cases in which emetine alone was used, the mortality rate was 5.5 per cent (Graph 9). As suggested in our previous publications[3, 47] on amebic hepatic suppuration and as a result of our personal experience in pleuropulmonary complications of this condition, we are of the opinion that emetine should be

administered in grain doses daily until 6 to 10 grains (0.39 to 0.65 gm.) have been given. According to Leake[59] the amount of emetine administered over any given period of time should not exceed 10 milligrams (⅙ gr.) per kilogram of body weight. A maximum dose for a person weighing one hundred and fifty pounds would be approximately 10 grains. Emetine should be used cautiously, because as shown by Rhinehart and Anderson,[60] working in Leake's laboratory, it produces in the experimental animal severe injury to the cardiac muscle. Similarly, as in uncomplicated amebic abscess of the liver, external open drainage is undesirable in nonsecondarily infected amebic infections of the pleura



Graph 10.—Graph showing incidence of recoveries in pleuropulmonary amebiasis in authors' series according to operative and nonoperative therapy.

and lung. Although theoretically the possibility of secondary infection of a hepatic abscess as a result of communication with a bronchus is possible, the danger of such a complication occurring if prompt therapy is instituted is relatively slight as evidenced by the results obtained by conservative therapy. In the collected cases the mortality rate was much higher when open drainage was used (48.2 per cent) than it was when emetine alone was used (5.5 per cent). When open drainage was combined with emetine therapy, the mortality rate was 15.3 per cent. The undesirability of operating upon patients with pleuropulmonary amebiasis is illustrated by the results obtained in our cases in which a 33.3

per cent recovery incidence was obtained in the group which was operated upon as contrasted with 100 per cent recovery incidence in the group which was treated conservatively with amebicides (Graph 10).

The necessity of evacuating an hepatic abscess or a pleural effusion resulting from the rupture of an hepatic abscess into the pleural cavity cannot be overemphasized. In those cases, however, in which there is a free communication between the hepatic abscess and a large bronchus, evacuation of the contents of the abscess usually occurs spontaneously. If the evacuation is incomplete, however, it is essential that the hepatic abscess be treated as an uncomplicated one, viz., by aspiration combined with emetine administration. Similarly in those cases in which perforation of the hepatic abscess into the pleural cavity has occurred, aspiration without open drainage should be done unless complicated by secondary infection. Similarly, as we[3, 47] have previously suggested in the treatment of amebic abscess of the liver an immediate smear of the pus should be made at the time of aspiration to determine the presence of microorganisms. If bacteria are found, especially if there is leucocytosis and hyperpyrexia, external drainage of the pus should be done because of the secondary infection; otherwise, aspiration alone should be done. Pellé[61] advocates the use of pneumothorax as an aid in preventing or controlling hemorrhage. This procedure may be an especially valuable adjuvant in those cases in which there is a large cavity and in which collapse is desirable, but the procedure can be used in relatively few cases because of the pleural adhesions which are usually present. Pellé[61] also suggests postural and bronchoscopic drainage. Thompson[32] advocated immediate operation in those instances in which the abscess failed to drain through the bronchus and in cases in which the pleural cavity was involved. He advised evacuation of the lung and liver abscess at the same time the pleural cavity was drained. Vergoz and Hermenjat-Gerin[36] advocate open drainage in those cases and immediate examination to determine whether microorganisms are present or not. If none are present, they advocate primary closure of the wound; otherwise, open drainage. We believe that the open evacuation of the abscess contents is not only unnecessary but also undesirable because of the greater danger of contamination. We cannot agree with Miginiac[55] that operation should be done in all cases of amebic abscess of the lung or empyema. As mentioned above, although the possibility of the contamination of the liver abscess through a hepatobronchial fistula is possible, apparently this rarely occurs, as evidenced by the statistics obtained in our collected and personal cases. We agree heartily with Craig[2] that many, if not most, cases of amebic infections of the lung will respond to emetine therapy alone and that relatively few will require surgical intervention. In the majority of the latter, simple aspiration of the originating focus in the liver will suffice.

Results.—In the present investigation in which 153 collected cases of pleuropulmonary amebiasis were analyzed, the cases were divided into five different groups according to the type of pleuropulmonary amebic infection:

1. *Hematogenous Pulmonary Abscess Without Liver Involvement.*—There were 22 cases in this group, of which only 1 (4.5 per cent) died (Table II, Graph 7). Although it would seem that the mortality rate should be high in this group, because in hematogenous pulmonary abscess amebas must be circulating in the blood stream, the low mortality rate obtained was undoubtedly due to the fact that all except 2 patients received emetine. One of those not receiving emetine was the fatal case mentioned above and another was a patient operated upon, but who recovered. One other patient was operated upon, but received emetine. In our series of cases there were no hematogenous pulmonary abscesses. The importance of emetine administration is well illustrated in this group, because even though a blood-borne infection or an amebiemia must be accepted, the mortality rate of 4.5 per cent is exceptionally low, undoubtedly due to the high incidence of emetine administration.

2. *Hematogenous Pulmonary Abscess and Independent Liver Abscess.*—There were 16 cases of hematogenous lung abscess occurring concomitantly with a liver abscess, the two, however, being unassociated (Table II, Graph 7). In the 16 cases there were 13 deaths (81.2 per cent mortality). Of the 13 fatal cases, 9 patients were operated upon, only 2 of whom received emetine. Three of the other 4 fatal cases had no therapy; one, however, received emetine. Three patients recovered, all of whom received emetine and 1 of whom was operated upon. Of the entire 16 cases, only 6 patients received emetine, 3 of whom recovered and 3 died. One of the former was operated upon; whereas 2 of the fatal cases were also operated upon. The high mortality rate in this group (81.2 per cent) as contrasted with the low mortality rate (4.5 per cent) in those cases of hematogenous lung abscess not associated with liver abscess is probably due to two factors. Even though in both groups of cases an amebiemia was present, in those cases in which there was a concomitant liver involvement the patient was undoubtedly more toxic because of the additional focus. The more important reason, to our minds, is the fact that relatively few, less than a third of the patients, in whom a liver abscess and hematogenous lung abscess occurred concomitantly received emetine as contrasted with 90 per cent of those patients in whom a hematogenous lung abscess alone was present. In our series of cases there were none in this group.

3. *Pulmonary Abscess Extending From a Liver Abscess.*—This comprised the largest group of the collected series. There were 58 cases, 25 of which (43.2 per cent) were fatal (Table II, Graph 7). Of the 25 fatal cases, 13 were operated upon. In 11 no therapy was used or the details

were incomplete and 1 received emetine alone. Of the 33 patients who recovered, 21 were operated upon, 4 of whom received emetine. The 6 additional patients who recovered received emetine alone. In 5 no statement was made concerning the therapy. In 1 postural drainage alone was used. Of the 11 patients who received emetine only, 10 recovered and only 1 died. Of the 58 cases there were only 42 in which details were given as regards therapy. Of these, 11 received emetine with 90.9 per cent recoveries. Of the 31 patients not receiving emetine, 13 died and 18 recovered (58 per cent). Of 34 patients operated upon, 13 died and 21 recovered (61.7 per cent). The effect of the administration of emetine is well illustrated by these cases, a 90 per cent recovery incidence in those cases in which emetine was given as contrasted with 58 per cent incidence

TABLE III

RESULTS IN AUTHORS' SERIES

TYPE OF THERAPY	PULMONARY ABSCESS EXTENDING FROM LIVER ABSCESS		BRONCHO-HEPATIC FISTULA WITH LITTLE PULMONARY INVOLVEMENT		EMPYEMA EXTENDING FROM LIVER ABSCESS		TOTAL	
	RE-COVERED	DIED	RE-COVERED	DIED	RE-COVERED	DIED	RE-COVERED	DIED
Operation					A 15	A 7	1	1
Operation and amebicides						A 8		1
Emetine or other amebicides	A 2, A 4, A 5, A 6, A 13, A 14		A 3, A 11, A 12		A 1		10	
No statement made or insufficient data		A 9			A 10		1	1
Total	6	1	3		3	2	12	3
	7		3		5		15	

in the group in which emetine was not given. The value of conservative therapy is also illustrated by the results obtained in our cases of pulmonary amebic abscess secondary to liver abscess rupture. Seven patients were of this type, 6 of whom received amebicides, all of whom recovered (Table III). Only 1 who had no treatment died.

4. *Bronchohepatic Fistula With Little Pulmonary Involvement.*—In the collected cases there were 30 patients with liver abscess and bronchohepatic fistula in whom the liver abscess evacuated through the bronchus with a minimal amount of pulmonary reaction (Table II, Graph 7). Of the 30, 27 recovered (90 per cent) and 3 (10 per cent) died. Two of the 3 fatal cases were operated upon. In 10 of the 30 cases insufficient data concerning the type of therapy were given. Emetine was administered to 10 of the 27 patients who recovered. Three of these were also operated

upon. All the patients receiving emetine recovered. Two of the 13 patients operated upon died, neither of whom had received emetine. Thus it is seen that half of the cases in which sufficient data are available received emetine, and all of these patients recovered. The only death in the series occurred in that group of patients who were operated upon and who did not receive emetine. There were 3 patients of this type in our series, all of whom recovered following the conservative treatment with amebicides (Fig. 8). It is obvious that bronchohepatic fistula with complicating amebic liver abscess with adequate evacuation of the abscess through the bronchus offers a relatively good prognosis and especially if emetine is administered.

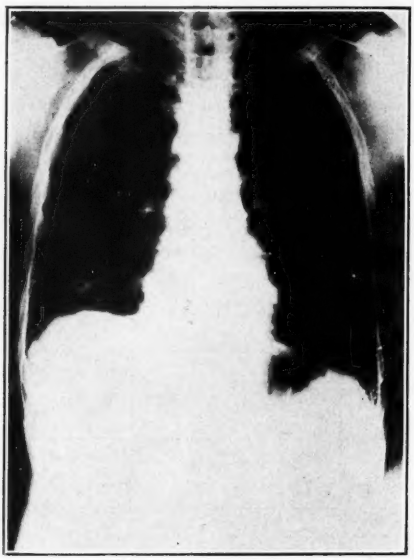


Fig. 8.—Anteroposterior roentgenogram of Case A 12 (shown in Figs. 2, 3, 4, 5). This plate was made approximately six weeks after those shown in Figs. 4 and 5, showing almost complete disappearance of shadow at base of right lung. The patient was symptom free at the time this roentgenogram was taken.

5. *Empyema Extending From Amebic Liver Abscess.*—In the series of collected cases there were 27 with liver abscess in which the abscess ruptured into the pleural cavity with the development of an empyema. In this group there were 21 deaths (77.7 per cent) (Table II, Graph 7). In 16 of the 21 fatal cases no details concerning therapy were given. Ten of these were observed early, before 1900. The other 5 fatal cases were operated upon. Of the 6 patients who recovered, 4 were operated upon, 1 of whom received emetine and 1 not operated upon also received emetine. Only 2 received emetine, both of whom recovered. In our series (Table III) there were 5 with empyema complicating amebic hepatic abscess. In 1 the pleural cavity alone was involved (Case A 7) and in

4 the pleural cavity and lung were involved concomitantly. Two (40 per cent) of the 5 patients died. Three (60 per cent) of the 5 were operated upon, 2 of whom died. Only 1 of the 5 patients received amebicides; he also was operated upon and died. These figures would indicate that the prognosis in empyema extending from amebic liver abscess is grave. On the other hand, the high mortality rates obtained in the collected series (77.7 per cent) and in our series (40 per cent) are probably due to the fact that emetine was used in few instances (20 per cent in both the collected and our series) and that open drainage was frequently employed (90 per cent in the collected cases and 60 per cent in our series). It is our prediction that had open operation been used less frequently and emetine administered more frequently, the mortality rates would have been much lower. It is important, however, that in these cases evacuation of the pleural contents be accomplished by aspiration in the noninfected cases and by some type of drainage in those cases which are secondarily infected.

The results obtained by the various methods of treatment in pleuropulmonary complications of amebiasis vary considerably (Table II, Graph 9). Of the 153 collected cases, 63 patients died (41.1 per cent). Of the entire group there were 47 cases in which insufficient data were given or in which nothing was done and in which no conclusions can be drawn concerning the results obtained by various therapeutic measures, of which 32 patients (68 per cent) died. Fifty-six patients were treated by open drainage alone, of which 27 (48.2 per cent) died. Twelve patients were treated by open operation and emetine, of whom 2 died (16.6 per cent). Thirty-seven patients were treated by emetine alone, of whom 2 (5.4 per cent) died. Of the entire group of 153 cases, 49 patients received emetine with only 4 deaths (8.1 per cent); whereas 59 of the remaining 104 died (56.7 per cent). The results obtained in these cases demonstrate that operation offers relatively little in the treatment of pleuropulmonary amebiasis. The mortality rate in this group is extremely high (48.2 per cent), but can be definitively decreased (16.6 per cent) by the administration of emetine. The best results, however (5.4 per cent mortality), were obtained in those patients who were not operated upon, but in whom emetine alone was used. In our 15 cases, 3 patients died (20 per cent). Of the 15 cases only 14 patients were treated, 1 dying before treatment could be instituted. Of these 14, 3 were operated upon, 2 (66.6 per cent) of whom died. Of the remaining 11 treated conservatively, none died (Graph 10). In our series there were 6 patients who received emetine alone or in combination with other amebicides; all of whom recovered. Seven received amebicides other than emetine, with one death. Two received no amebicides, both of whom died. As mentioned under treatment, we are of the opinion that emetine is the specific drug in the treatment of pleuropulmonary amebiasis.

SUMMARY AND CONCLUSIONS

1. An analysis of 153 collected and 15 of our own cases of pleuropulmonary amebiasis is presented.

2. Pleuropulmonary complications of amebiasis occur much more frequently than is generally supposed. They occurred in 15.8 per cent of 2,490 reported and in 15.7 per cent of our 95 consecutive cases of amebic hepatic abscess.

3. The greatest number of patients in the collected series (38 per cent) were in the third decade of life; whereas the greatest number in our series (40 per cent) were in the fifth decade of life.

4. Males are involved much more frequently than females (96.2 per cent in the collected and 93.3 per cent in our series). In our series 9 patients were white and 6 colored.

5. Pleuropulmonary complications of amebiasis occur, as a rule, resulting from an extension of amebic hepatic abscess. Perforation of the abscess rarely occurs into the free pleural space, but more frequently into the lung or bronchus. Exceptionally hematogenous pulmonary amebic abscesses may occur.

6. Pleuropulmonary amebic infections are classified into five groups, depending upon the type of pleuropulmonary involvement: (1) hematogenous pulmonary abscess without liver involvement; (2) hematogenous pulmonary abscess and independent liver abscess; (3) pulmonary abscess extending from liver abscess; (4) bronchohepatic fistula with little pulmonary involvement; (5) empyema extending from liver abscess. In 14.3 per cent of the collected series the pleuropulmonary lesion was a hematogenous pulmonary abscess without liver involvement. In 10.4 per cent the lesion was a hematogenous pulmonary abscess together with an independent liver abscess. In 37.2 per cent the lesion was a pulmonary abscess extending from a liver abscess. In 19.6 per cent the lesion was a bronchohepatic fistula with little pulmonary involvement, and in 17.6 per cent the lesion was an empyema extending from a liver abscess. Pulmonary abscess extending from a liver abscess comprised 46.6 per cent of our 15 cases, bronchohepatic fistula with little pulmonary involvement, 20 per cent, and empyema extending from a liver abscess, 33.3 per cent.

7. The clinical manifestations of pleuropulmonary amebiasis consist chiefly of cough and expectoration, fever, diarrhea, enlarged and tender liver, pain in the chest, and cachexia. Cough and expectoration were observed in 92.5 per cent of the collected cases, fever in 43.2 per cent, a history of a previous diarrhea was obtained in 41 per cent, an enlarged liver present in 39.7 per cent, diarrhea at the time of admission in 33.5 per cent, and pain in the chest in 31.3 per cent. The expectoration of chocolate sauce pus is indicative of a communication between a

liver abscess and a bronchus and is of diagnostic importance. Pulmonary manifestations consist of consolidation and cavitation.

8. There is characteristically a moderate leucocytosis without concomitant increase in the polymorphonuclear leucocytes. A marked leucocytosis is indicative of a secondary infection.

9. X-ray examination shows elevation and fixation of the diaphragm and a shadow at the right base, particularly in those cases in which a pulmonary abscess extends from a liver abscess. Characteristically the shadow is triangular with the base below and the apex above.

10. Diagnosis can be definitively established by the characteristic chocolate sauce pus and the finding of amebas in the sputum and aspirated material.

11. The prognosis in pleuropulmonary amebiasis is dependent upon the type of the lesion and the therapy employed. The mortality rate in the collected series was extremely high (41.1 per cent). In the group with liver abscess and hematogenous lung abscess the mortality rate was 81.2 per cent. In the cases of liver abscess with empyema it was 77.7 per cent. In cases of lung abscess extending from liver abscess it was 43.2 per cent. In cases of bronchohepatic fistula with a minimal amount of pulmonary involvement it was 10 per cent. In the cases of hematogenous lung abscess without liver abscess it was 4.5 per cent. The prognosis depends probably more upon the type of therapy than upon the type of lesion. The mortality rate obtained in the cases treated by open drainage was 48.2 per cent, that in the cases treated by open drainage and emetine, 16.6 per cent, whereas that obtained by emetine alone was 5.4 per cent.

12. The treatment of pleuropulmonary amebiasis consists of the administration of emetine and the aspiration of those abscesses not sufficiently evacuated through the bronchus. Open drainage should never be done except in those cases with secondary infection. The importance of emetine in the treatment of amebic pleural pulmonary complications is illustrated by the results obtained in the collected and in our cases. The incidence of recovery in cases treated without emetine in the collected cases was 43.9 per cent and in our cases 40 per cent; whereas in those cases receiving emetine the respective incidences were 91.8 per cent and 100 per cent.

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DISCUSSION

DR. FRANK K. BOLAND.—I was interested to hear Dr. Ochsner say that he found the ameba in the pus and sputum in the majority of these cases. That has been contrary to our findings in amebic abscess of the liver. Of twenty cases of amebic abscess of the liver, we have found amebas in the pus in only two cases. We usually found amebas in the scrapings from the wall of the abscess cavity, but in the pus in only two cases among the twenty. So it would seem that it is easier to find amebas in the sputum and pus in these thoracic cases than it is when the lesion is confined to the liver. We find amebas in cases of amebic dysentery in almost a hundred per cent, but it always struck me that as soon as there were complications of amebic disease, such as abscess of the liver and ulcers of the colon, the ameba seemed to disappear from the discharges.

DR. HARRY BALLON.—I should like to ask Dr. Ochsner two questions: first, in the absence of amebas in the sputum or stool, what response to emetine therapy has he noted in patients whose lung abscesses are otherwise suggestively amebic in origin; second, how many of the patients referred to in his presentation gave clinical histories of long standing, of six months to a year or more?

DR. ALTON OCHSNER.—As regards the finding of amebas in the pus obtained from hepatic abscesses, our experience in New Orleans has been similar to that of Dr. Boland in that amebas are seldom found in the pus obtained from hepatic abscesses, but usually can be demonstrated in the scrapings of the abscess wall. This is probably due to the fact that the amebas in the cavity of the abscess have died.

In answer to Dr. Ballon's question, I am of the opinion that in most of our cases the extension of the amebic infection from the liver to the lungs has been relatively recent.

We are convinced that amebic infections are not interesting, but that people are becoming more conscious of the infection and for this reason the diagnosis is more frequently made than previously. We believe that amebiasis of all types and especially the pleuropulmonary types occur more frequently than is commonly supposed.

The response of pleuropulmonary amebiasis to emetine therapy is dramatic. There is probably no thoracic condition which responds so well to therapy as do these lesions. We are inclined to believe that those reported cases of lung abscess which have been successfully treated by emetine are amebic in origin, although supposedly they are not the result of amebic infection. I, personally, doubt whether emetine has any value in the treatment of nonamebic pulmonary suppurations.

The danger of emetine must, however, be emphasized. Emetine is a dangerous drug, because it is a muscle poison and will produce myocardial damage if not used cautiously. We employ emetine in grain doses daily until ten doses are given.

Dr. Blalock has referred to the prophylactic treatment of pulmonary embolism. For the past three years we have been using leeches in the treatment of thrombophlebitis, and believe from the clinical results which we have obtained that this form of therapy has some value prophylactically in pulmonary embolism. Following the application of leeches to an extremity of a patient with thrombophlebitis, the pain which the patient has previously complained of usually is relieved within an hour's time, and, as a rule, within eight to ten hours after the application of the leeches the redness disappears. In many cases elevation of temperature subsides after twenty-four hours. We feel encouraged from our clinical results and believe that the use of leeches in thrombophlebitis has definitely shortened the convalescence and possibly has prevented cases of pulmonary embolism.

BRONCHOGRAPHY FOLLOWING THORACOPLASTY FOR TUBERCULOSIS*

HENRY L. CABITT, M.D., J. J. SINGER, M.D., AND
EVARTS A. GRAHAM, M.D.
ST. LOUIS, MO.

THE purpose of a thoracoplasty in tuberculosis is to create a sufficient degree of collapse or compression of the lung to aid in the promotion of healing of the lesions. If a cavity is present, the desire of the surgeon is to obliterate that cavity by the operation. If, however, a cavity remains unobliterated after the lapse of sufficient time for the maximal postoperative contraction of scar tissue, in one sense the operation has been a failure. Cavities, however, are not the only lesions compression of which improves the patient's condition. Tuberculous bronchiectasis can give rise to the expectoration of sputum containing tubercle bacilli, and several of our patients with this condition have been benefited by thoracoplasty. It is likewise true that compression of the tissue around a cavity is of great assistance in diminishing the symptoms of the patient even if the cavity itself is not completely obliterated by the operation.

It is appropriate at this time perhaps to diminish the amount of emphasis on operative mortalities and to concentrate more attention upon the results of our thoracoplastic operations. By this assertion is meant also that in our opinion we should not be content with the statements of the patients in regard to their ability to work, the characteristics of the sputum, etc., but that we should seek to determine how far our operative results have approached, in an anatomical sense, the object for which the operation has been undertaken. There can probably be no dispute about the statement that the more nearly complete the compression of the diseased part of the lung is, the better will be the clinical result. Atelectasis has long been recognized as a benefit to a patient with tuberculosis. The conscientious surgeon, however, seems always to be in a dilemma in deciding, on the one hand, how much of an operative procedure will be required to accomplish the desired objective and, on the other hand, how suddenly the cavity of a patient can be obliterated without the harmful results of a compression that may be too rapid. The surgeon who is constantly seeking to improve the anatomical results of thoracoplasty may therefore have a relatively high operative mortality, but a critical study five years later of the patients

*From the Medical and Surgical Chest Service of Barnes Hospital, and Washington University School of Medicine.

Read at the Eighteenth Annual Meeting of the American Association for Thoracic Surgery at New York City, June 3, 4, and 5, 1935.

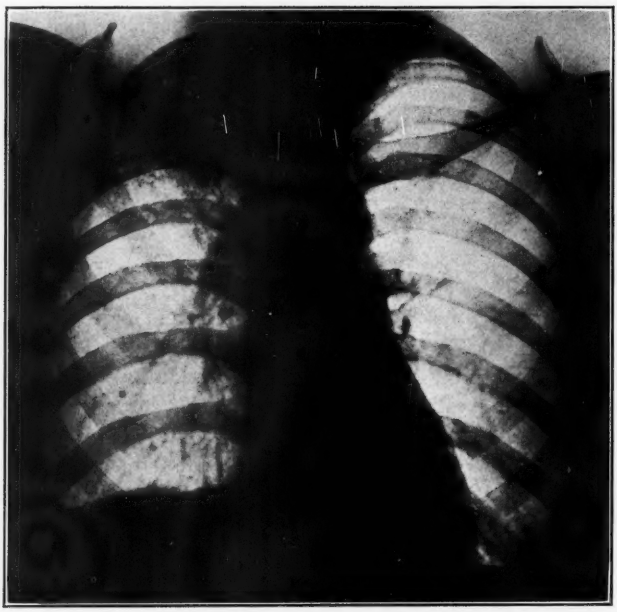

Fig. 1*A*.—H. J., Nov. 22, 1934. Film before operation shows right apical involvement. The rest of that lung and also the contralateral lung appear normal.

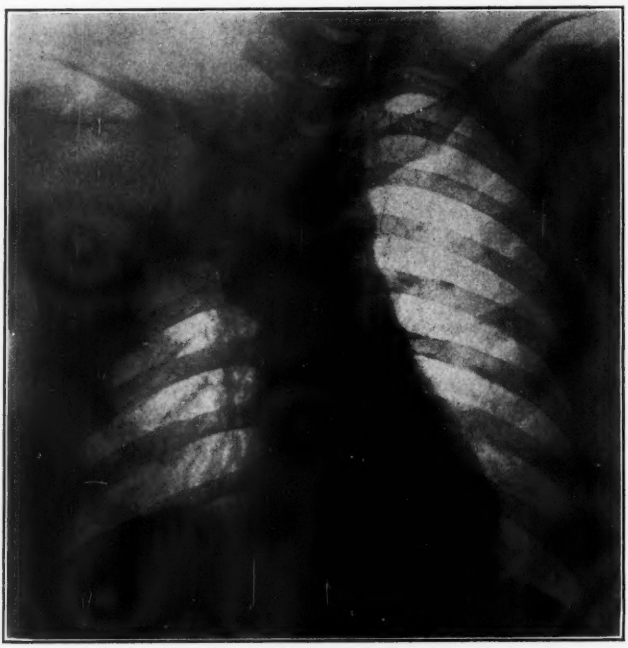

Fig. 1*B*.—Jan. 28, 1935. A collapse of the right upper lobe is shown as a result of an upper thoracoplasty, but the condition of the bronchi cannot be visualized.

upon whom he has operated probably will show a greater percentage of healed cases than will a similar study made upon the patients operated upon by an overcautious surgeon who is more intent on his operative mortality than on his anatomical results.

The purpose of this communication is to recommend a procedure which we have found helpful in a more accurate evaluation of our results after thoracoplasty for tuberculosis than has been possible without this method. In an attempt to determine more accurately for our own satisfaction the condition of the lung after operation, we have subjected twenty patients to examination with

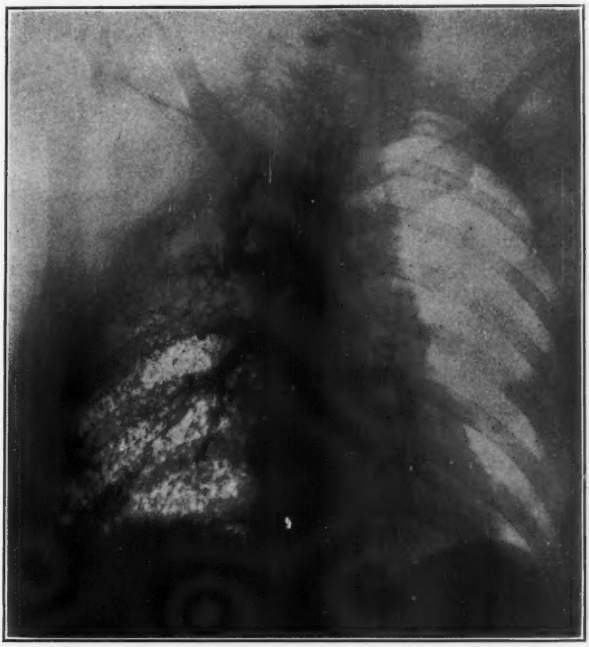

Fig. 1*C*.—Jan. 29, 1935. The lipiodol instillation has resulted in a clear demonstration of the upper lobe bronchus. The main bronchus of the upper lobe is seen satisfactorily collapsed, and only small bronchi are visible. We interpret this finding as indicative of a satisfactory anatomical result. The clinical result was equally satisfactory.

lipiodol after thoracoplasty. We make no claims for priority in the use of lipiodol for this purpose. Neuhof[1] last year reported a rather extensive use of lipiodol in the examination of patients with pulmonary tuberculosis and in some cases after thoracoplastic operations. We desire to report our experience with these twenty patients chiefly because we feel that the method is one which should be utilized more extensively in attempts to determine just what the postoperative conditions are. It would seem that by utilizing such a procedure the

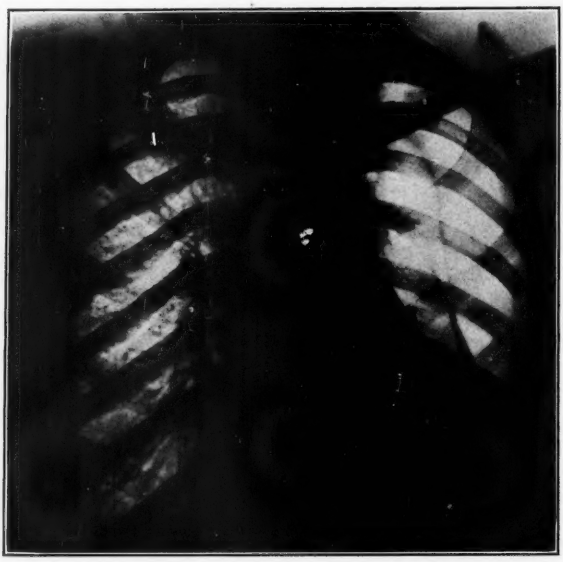

Fig. 2*A*.—S. E., Nov. 27, 1934. Film made before thoracoplasty shows left pneumothorax with partial collapse of lung.

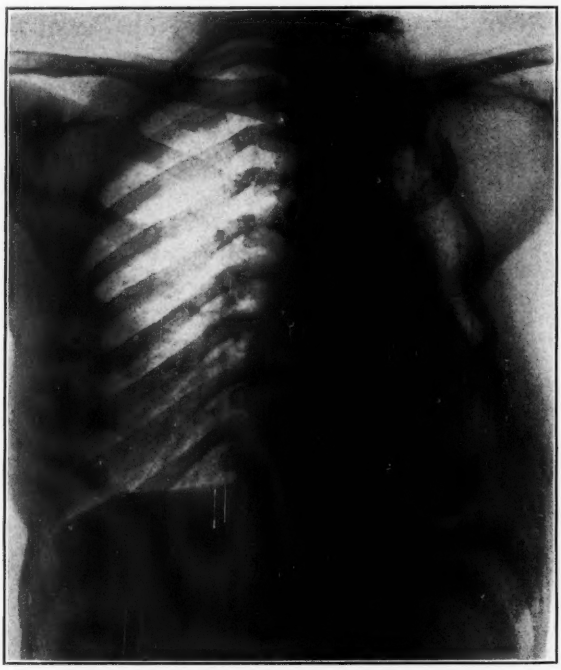

Fig. 2*B*.—Jan. 16, 1935. After a thoracoplasty of the upper seven ribs the bronchial tree is not evident, and there is still a small amount of pneumothorax present.

prognosis can be determined with somewhat greater certainty. There is still present in the minds of many physicians who deal with tuberculosis the fear of harmful effects following the use of lipiodol in tuberculosis. For several years, however, we have used it freely and have seen no serious effect. Singer and Francis,[2] in 1929, published a report on the investigation of thirty-five cases of pulmonary tuberculosis by bronchography with no ill consequences.

In evaluating the amount of compression following thoracoplasty it has been the usual custom to note by an ordinary x-ray examination what changes have taken place in the lung. A homogeneous shadow in

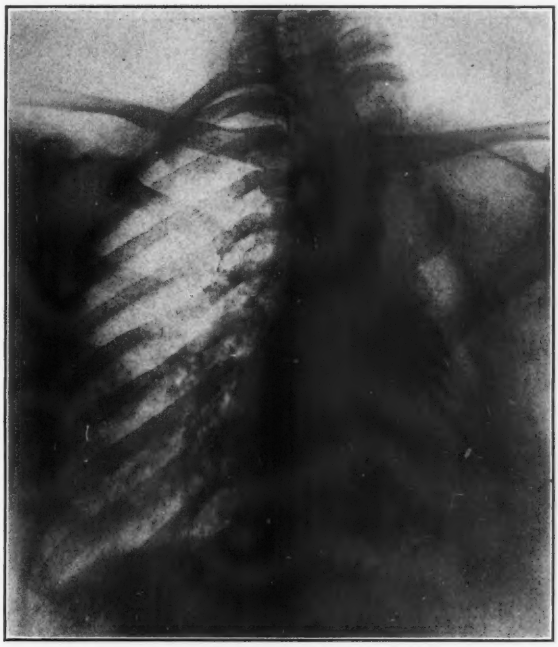

Fig. 2C.—Jan. 22, 1935. The lipiodol film shows the bronchi to be well collapsed and no evidence of cavity. This is interpreted as a satisfactory anatomical result which agreed with the satisfactory clinical result.

the film with evidence of marked narrowing of the corresponding side of the thorax has been generally regarded as evidence of satisfactory compression. Nevertheless, it has been the experience of ours and of others that many patients who present such a homogeneous shadow may still expectorate a considerable quantity of sputum containing tubercle bacilli. In such cases the instillation of lipiodol has been of very great value because often the cause of the persistence of sputum has been revealed by that means. In several instances a tuberculous bronchiectasis has been shown in this manner and a reoperation with the removal of

all of the remaining portions of ribs in order to increase the compression has resulted in a marked diminution of the amount of sputum. At any rate, the instillation of lipiodol has enabled us to feel a little more certain concerning the probability of success or failure in the operation's accomplishing the desired result.

The method of instillation of the oil for bronchography which we have used was the one described by one of us (J. J. S.) nine years ago. We feel that this is the simplest of the various methods employed. The patient is placed in a good light, either natural or artificial. He is instructed to breathe deeply and not to swallow or cough during the

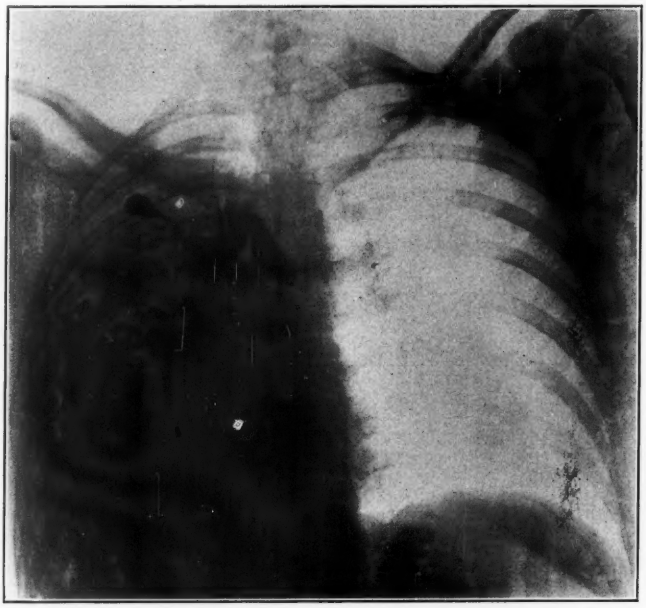

Fig. 3A.—N. G. Lipiodol film before thoracoplasty shows extensive tuberculous bronchiectasis.

instillation of the oil. The tongue is gently but firmly grasped by the operator. The oil, which has been previously warmed, is slowly injected so that the stream strikes the base of the tongue. No anesthesia is required except in unusual cases; then only cocainization of the pharynx is employed. In children the method is also applicable if cocainization of the nose and pharynx is done and a nasal tube used. After 20 c.c. of the lipiodol has been injected, the patient is instructed to lean to one or the other side so that the oil will flow of its own weight into that lung or that portion of the lung which it is desired to study. In those cases in which the upper portion of the lung is being

studied the patient is placed on his back after all the oil has been injected. In that way the upper bronchi can usually be outlined if the films are made with the patient in that position before a cough or any other movement will permit the oil to flow into the opposite lung. The films are made in such a way as to show the denser shadow rather than the soft parenchyma of the lung. The patient is then instructed to cough up all the oil he can. The small amount which is not expectorated is usually rapidly eliminated. In only one of our cases has there been any evidence of iodism and in that case it was minimal.

It is common knowledge, of course, that a patient may still have tubercle bacilli in his sputum, although the anatomical conditions pro-

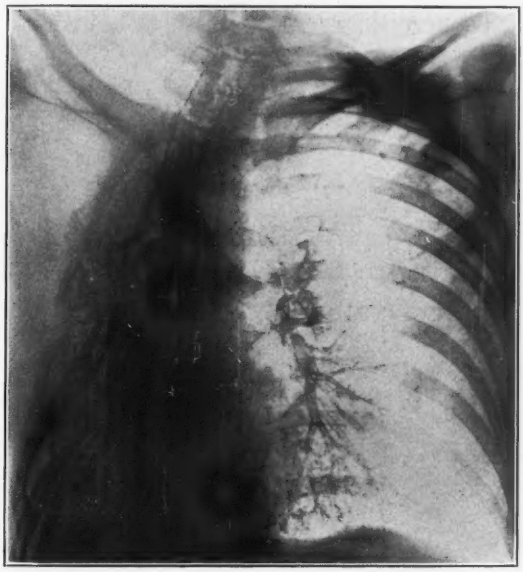

Fig. 3*B*.—Lipiodol film after thoracoplasty shows the bronchiectatic dilatations to be rather satisfactorily collapsed.

duced by a thoracoplasty may seem to be thoroughly satisfactory. Even if a lipiodol examination fails to reveal any further evidence of uncollapsed cavity or bronchiectasis, occasionally the sputum may still be positive. It is recognized, of course, that surgical collapse cannot of itself cure tuberculosis. All that it can be expected to do is to make the conditions present more suitable for the healing of the tuberculosis by natural processes. It is, therefore, a common experience that patients with positive sputums will subsequently fail to show tubercle bacilli in their sputums after a sufficient time has elapsed for the adequate contraction of scar tissue which probably is important in the complete obliteration and healing of any small cavities which may be present.

Again, of course, one can never be certain whether the tubercle bacilli which are present may be coming from the collapsed lung or from some minimal lesion in the other lung. If, however, a lipiodol examination of the patient after a thoracoplasty reveals that adequate anatomical collapse has been accomplished, the probabilities are greater that in time the sputum will become negative and that the patient will become an arrested case than if such an examination reveals the presence of cavitation or bronchiectasis still remaining.

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SURGICAL REVISION OF UNSATISFACTORY THORACOPLASTY BY REOPERATION AND EXTRAPERIOSTEAL (SUBSCAPULAR) PACKING*

THOS. J. KINSELLA, M.D., F.A.C.S.
OAK TERRACE, MINN.

THE ideal thoracoplasty in the treatment of pulmonary tuberculosis should produce a complete mechanical obliteration of cavity or empyema pocket and be followed by permanent disappearance of all symptoms both toxic and local. Unfortunately this result is not always accomplished, much to the chagrin and discomfiture of the surgeon. While the causes of such failure may be altogether beyond the surgeon's control, he is frequently blamed for it and is eventually confronted with the none too pleasant problem of attempting to improve the result.

The question of what constitutes an unsatisfactory thoracoplasty is one concerning which considerable controversy may easily be developed. Certainly there are a number of patients whose results fall somewhat short of the ideal mentioned above who may be, and not infrequently are, successfully rehabilitated. Such patients, if conscientious and well trained, may present little or no public health problem even though from time to time they do raise small amounts of sputum containing tubercle bacilli and may continue to perform useful work for many years. Potentially such patients may be less of a menace to those around them than to themselves, for they are confronted with the ever present danger of spreading tuberculosis into new areas of the lung either on the same or on the contralateral side.

The problem of deciding what constitutes an unsatisfactory thoracoplasty, strange as it may seem, is not always an easy one. It must be considered from several angles before a final conclusion is reached. Theoretically, uncollapsed cavity associated with positive sputum following thoracoplasty should be prima facie evidence of an unsatisfactory result, but practically the problem is not always so simple. While ideally we prefer to have all of the patient's cough and expectoration disappear promptly following the thoracoplasty series, experience teaches us not to expect such good fortune in every instance. The reason for this is quite obvious, for if we consider the appearance of the lining wall of the tuberculous cavity, we are in-

*From the Departments of Surgery, Glen Lake Sanatorium, Oak Terrace, and University of Minnesota, Minneapolis.

Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery, New York, June 3, 4, 5, 1935.

clined rather to marvel that any of the patients become symptom-free in a short period of time. When cough and expectoration do abate suddenly, it is well to be certain that this is not due to mere retention rather than cessation of secretion. Not infrequently the amount of sputum is increased temporarily, following surgical collapse, to be followed by alteration of character, then decrease in amount and subsequent disappearance of all expectoration. A number of weeks must elapse in some patients before this occurs, even when everything else points to a completely satisfactory result. Apparently this is brought about not entirely by the collapse produced by the thoracoplasty itself, but occurs with the healing of the tuberculosis from the immobilization resulting from the operative procedure. Likewise in some individuals all sputum containing tubercle bacilli may disappear very promptly and remain absent for weeks or months, to reappear subsequently either with or without demonstrable evidence of cavitation. Again, all sputum from the original area of tuberculosis in the collapsed lung may subside but some expectoration still persist, the material coming from an associated bronchiectasis or even from a contralateral focus.

There is at times considerable divergence of opinion as to what constitutes a positive sputum. It is a well-recognized fact among tuberculosis workers that certain patients raise lung sputum only at certain times of the day, and some only periodically. Single specimens, therefore, may give a false impression of conditions while a twenty-four-hour sputum collection may tell a different story. The concentration method of sputum examination theoretically at least should give a higher percentage of positive results than the ordinary routine method. Guinea pig inoculations or cultural methods provide a much more delicate mechanism for finding tubercle bacilli than a simple microscopic examination of a stained smear, a relatively few tubercle bacilli being necessary to produce a culture or kill a guinea pig whereas approximately 100,000 bacilli per cubic centimeter are necessary to be discovered under the microscope as Gaffky I. Guinea pig inoculations with stool material will at times disclose the presence of tubercle bacilli in swallowed sputum when the patient reports that he is raising no sputum whatsoever. From this it may be readily seen that a result which is satisfactory today may become unsatisfactory later and sputum which might be considered negative in one clinic may be considered positive in another.

The demonstration of cavity in the collapsed lung following thoracoplasty should prove an incomplete or unsatisfactory result theoretically, at least. Physical examination, which is notoriously inaccurate in demonstrating certain types of cavity in the uncollapsed lung, is rendered even less reliable following surgical collapse, by alteration in transmission associated with the increased density of the lung from

collapse, and the more intimate association of trachea and primary bronchi with the chest wall. It can, therefore, not be relied upon to prove or disprove the presence of uncollapsed cavity following thoracoplasty, particularly with small excavations or where there is really a serious question of doubt.

The x-ray examination offers our most valuable single diagnostic method. It is useful in two ways: first, to determine the presence or absence of cavity; and, second, to aid in the localization of its position within the thoracic cage. X-ray films taken on ordinary exposure soon after extrapleural thoracoplasty may fail to reveal uncollapsed cavity in the underlying lung because of infiltration in the tissues, edema of the pleura, or accumulation of fluid in the subscapular space; whereas subsequent films a few weeks later, using the same technic, may visualize cavity without difficulty. Special stereoscopic x-ray films using a technic suitable for bone with the aid of the Bucky Potter diaphragm are of great aid in demonstrating and localizing cavities within the chest. Great care and considerable experience with chest films of thoracoplasty patients are necessary, however, for accurate interpretation of such films. In spite of the common tendency to assume that because a cavity is markedly reduced in size by surgery, it will become obliterated eventually by subsequent fibrosis or contraction, we note that all too frequently the converse is true. Cavity which is only partially obliterated, subsequently is more likely to become larger rather than smaller.

When we have once located the presence of cavity in the collapsed chest, it is advisable to attempt to determine its position within the lung before making further attempts at its closure. It is a recognized fact that the majority of tuberculous cavities lie in the posterior half of the chest prior to the institution of collapse therapy.[1] Some, however, lie anteriorly, while others occupy the whole apex of the lung or the whole upper lobe. Following thoracoplasty and the application of external compression anteriorly, cavities become displaced medially, posteriorly, and downward. If the cavity lies anteriorly, it may be displaced inward considerably with shift of the mediastinum following collapse and compression. If situated more posteriorly, following collapse of the chest wall it may come to lie more or less in the paravertebral gutter, apparently under cover of the transverse processes, frequently between the third and the sixth dorsal levels. From the practical standpoint these may lie not directly in relation to the transverse process but considerably anterior to it, depending somewhat upon the location of the original cavity. Determination of the exact position of cavity from stereoscopic x-ray films alone is difficult in the collapsed chest. Because of their relatively small size and the absence of fluid level as well as the associated infiltration of the chest wall and lung, lateral chest films do not give

so much help in localizing their relative position in the lung as in the case of nontuberculous lung abscess or cavity in the uncollapsed chest. A comparison of the relative size of such a cavity on posteroanterior and anteroposterior films may give some idea as to whether it lies closer to the back or to the front of the chest.

Some help in determining the position of smaller cavities prior to operation may be obtained from the following calculation obtained from a study of stereoscopic chest films, the position of the cavity being determined from the amount of vertical shift of the cavity on the two stereoscopic films as follows. The two stereoscopic films of the chest are superimposed so that the date and number markers exactly coincide. It will then be seen that a certain point on the cavity wall has undergone vertical displacement between the two films, the amount of which is determined by the relative distance of the cavity from the anterior chest wall on films taken in the posteroanterior direction. This vertical displacement may be measured and substituted for D in the following formula: $X = \frac{AD}{C+D}$, in which X equals the distance of the cavity from the anterior chest wall, A equals the distance from the tube target to the film, D is the vertical displacement of the cavity between the two stereoscopic films, and C is the vertical distance which the x-ray tube is shifted between the two films.[2] Knowledge of the position of the cavity in relation to the dorsal transverse processes as well as its relation to the anterior or posterior chest wall is essential before additional surgery is undertaken in an attempt to obtain its obliteration, in order that the surgical attack may be directed against the proper point in the chest wall overlying the cavity.

CAUSES OF UNSATISFACTORY THORACOPLASTY

There are a number of factors which contribute to the production of an unsatisfactory result following extrapleural thoracoplasty, no single one being responsible for all failures. Frequently two and at times even several of these factors may come into play in the production of an unsatisfactory result. Listed somewhat in the order of their frequency, these causes may be mentioned as follows:

1. Huge cavity.
2. Insufficient rib resection—because of
   - Number of ribs
   - Length of segments
   - Long posterior stumps

   (1) patient's condition
   (2) flexible chest wall
   (3) age and type of thoracoplasty.
3. Partial thoracoplasty.
4. Delay between stages:
   - Poor condition of patient
   - Spread of tuberculosis
   - Infection of wound
   - Poor cooperation.

5. Continued excavation:
   Incomplete excavation
   Late recurrence of cavitation.
6. Rigidity of chest wall and pleura:
   Thickened pleura
   Calcification of pleura.
7. Rigidity of lung:
   Infiltration
   Bone and cartilage in cavity wall
   Calcification of cavity wall.
8. Inefficient compression.
9. Inadequate postoperative treatment of tuberculosis.

1. The size and location of the cavity is probably of primary importance in the production of an unsatisfactory result. It is quite evident, theoretically at least, that a huge cavity, 10 cm. or more in diameter, occupying the whole upper portion of the chest should be closed with more difficulty than a smaller excavation lying well within the substance of the lung. When a cavity occupies the whole apex, little or no lung tissue remains in this area, and extreme collapse is necessary in order to bring the two walls into apposition. Any cavity 5 cm. or more in diameter may present a difficult problem. Closure of the first 75 per cent of any cavity is accomplished much more readily than the last 25 per cent.

2. Insufficient rib resection is probably the next most important factor in the production of an insatisfactory result following a series of thoracoplasty operations. The question of what is insufficient is of course entirely a relative one, as what might be entirely adequate for one patient may prove totally inadequate for another. When viewed from the standpoint of our present ideas of thoracoplasty where total deribbing of the upper chest, either as a primary or as a combination procedure, is frequently performed, we must admit that many of our thoracoplasties performed five years or more ago were inadequate, yet a surprising number of good results were obtained. Certainly no one today would willingly perform a Wilms-Sauerbruch type of thoracoplasty with its limited resection of the upper ribs and expect to obtain a high percentage of good results, especially in the presence of large cavity. Any surgical series which includes a number of these older cases will likewise present a number which eventually prove themselves unsatisfactory. This group is probably of greater importance now than it will be a few years hence. Certain conditions such as an extremely flexible chest wall or very flexible mediastinum may even now warrant limited rib resection. Likewise, changes in the condition of the patient necessitating termination of the operation as a matter of necessity rather than election may produce the same effect, leaving the patient with a limited rib resection or unnecessarily long vertebral stumps.

3. The increasing use of partial thoracoplasty operations is and probably will continue to be a factor in the production of late unsatisfactory results, the uncut lower ribs holding out the lung in the midportion. There is always a temptation to attempt to save the patient additional operations by omitting resection of the lower ribs as well as to be satisfied with nearly complete results for the time being in the hope of later improvement, rather than insisting upon more surgery at once. Not a few of these "almost complete results" will eventually require reoperation to obtain additional collapse. Twenty of the forty patients reoperated in this series had previously had partial thoracoplasty; while ten of the last fourteen patients reoperated were in the partial thoracoplasty group.

4. Certain conditions, largely beyond the operator's control, may arise which may seriously interfere with obtaining the desired collapse, by forcing the surgeon to wait beyond his ideal time interval between the various stages of thoracoplasty, notably, poor condition of the patient, exacerbation or spread of tuberculosis, infection of the wound, or poor cooperation from the patient. Poor condition of the patient is a factor which becomes of increasing importance as the field for thoracoplasty is broadened and more unfavorable cases are included in the group. Such individuals carefully handled may be successfully treated by thoracoplasty, but when treated the same as the ordinary individual in good condition might be, may well produce bad results or additional fatalities in the series. Spread of tuberculosis either in the same or in the contralateral lung demands delay before further surgery is undertaken and may be the immediate cause of an unsatisfactory result necessitating reoperation at a later date in an attempt to obtain more satisfactory collapse. The age of the patient is likewise important in this respect, rib regeneration being considerably more rapid in the younger individuals than in those of more advanced years. Infection in the thoracoplasty wound may force considerable delay between the various operations. In our experience this has been unusual and has not been a factor in this series. Delay between stages or an incomplete operation may result from poor cooperation on the patient's part either because of his general make-up or because he has been lulled into a false sense of security by marked improvement in his symptoms following an original operation even though a complete result has not been obtained and the cavity is not completely closed.

5. Thoracoplasty undertaken while the process of excavation is still in progress may produce what is apparently an excellent result immediately, but be followed later by the reappearance of cavity or an increase in the size of one incompletely collapsed. This may be the result of continued excavation of an area already more or less

caseous when the surgery was undertaken, the process of excavation continuing in spite of the collapse, with the resultant apparent reopening of the cavity at a later date. Reactivation of a tuberculous process, even years after the original collapse, may likewise produce cavity or recurrence of symptoms. From our experience it is quite evident that a cavity which is incompletely closed at the time of the original operation has a greater chance of increasing in size as time goes on than eventually of becoming obliterated.

6. Marked fibrosis or infiltration of the visceral and parietal pleura may very seriously interfere with collapse of the lung during thoracoplasty. This may range from a slight infiltration over an area of underlying disease to a marked induration or almost boardlike rigidity of the pleura infiltrated as a result of a chronic pyothorax. The extreme of this, calcification of the pleura following a purulent exudate, may produce a pleural shell which is absolutely unyielding.

7. Marked fibrosis of the lung itself as a result of a very chronic disease or marked infiltration which has not progressed to cavitation may mechanically interfere with the closure of a cavity and the production of desired results. It may be encountered either in single cavity or as more frequently happens in the presence of the honeycombing type of cavitation within a dense fibrotic lung. The cavity wall itself, in some instances, may be extremely rigid because of its own fibrous shell or even because of a lime deposit in its periphery or the growth of cartilage or bone in the wall. This presents a problem not easily handled at primary operation and possibly even more difficult at the time of secondary interference.

8. Collapse of the cavity or empyema pocket by thoracoplasty should be obtained by adequate rib resection and not by external pressure. There is a tendency for the chest wall to expand gradually to a certain degree before firm rib regeneration occurs with a resultant loss of some of the collapse originally obtained. I believe that it is possible by the early use of postoperative compression to obtain a better permanent collapse than in the same patient with the same operation without some form of external fixation.

9. It is a well-recognized fact among phthisiologists that surgery in itself does not cure the tuberculous lesion in the lung. It corrects mechanical difficulties and brings about conditions which favor the healing of the tuberculous process, but time, the patient's resistance, and bodily reaction against the disease are necessary to obtain a final result. Insufficient postoperative treatment of tuberculosis in itself predisposes to an incomplete healing of the tuberculous process in the collapsed lung and favors the continuation of a smoldering area of disease which may eventually result in recurrence of symptoms or reopening or enlargement of cavity.

METHODS OF SURGICAL REVISION

The actual operative procedure necessary must be determined in each individual patient by local conditions present, the size and location of the cavity, and by the type of thoracoplasty which was performed at the original operation. Simply expressed, this means a complete deribbing of the chest overlying the uncollapsed cavity extending well above and well below the involved area to permit more adequate collapse. It involves the removal of rib which was not taken at the previous operation, together with all regenerated rib in this same area. If the original thoracoplasty was of the very limited Wilms-Sauerbruch type in which the posterior ends of the anterior segments have migrated backward and approximated the transverse processes, anterolateral resection of the upper four or five ribs and their cartilages with removal of the regenerated segments posteriorly through an axillary approach may give the desired results. No patient whose difficulties could be conquered so readily has been included in this series. If the regenerated rib posteriorly is of greater extent or more irregular, it may be well to perform the operation in two stages, performing an anterolateral removal of the unresected anterior ribs and cartilages at the first stage, followed later by a posterior approach in which the regenerated ribs to the transverse process are removed. If additional surgery is indicated because of an incompletely collapsed tuberculous empyema pocket, containing a persistently purulent exudate, it may be necessary not only to remove all overlying ribs, but eventually to unroof the residual pocket and remove all caseous material which has prevented fusion of the visceral and parietal surfaces, packing lightly until healthy granulation appears before doing a secondary closure. Patients presenting such conditions are likewise not included in this report.

A considerable number of the patients who require reoperation because of uncollapsed intrapulmonary cavity have excavations which have receded inward so that on x-ray films they seem to lie inside the transverse process. Under such conditions simple reoperation with removal of all the uncut ribs plus regenerated ribs from sternum to transverse process may prove inadequate, particularly so if the chest wall is somewhat rigid, the underlying pleura thickened, or the lung itself infiltrated. Under such conditions, reoperation alone must frequently be supplemented by the application of some type of compression in order to produce a complete result. It is to such a combination of reoperation supplemented by the application of firm compression within the wound that we have gradually turned for a solution of this problem.

TECHNIC OF REOPERATION

The technic of anterolateral thoracoplasty needs no comments, having been adequately described previously by Welles[3] and Hedblom.[4] If reoperation posteriorly is performed within a few months of a posterior thoracoplasty which has included careful isolation and crushing of the upper intercostal nerves, the whole procedure may be readily performed under local anesthesia with a minimum of injection necessary in the chest wall. When a longer time has elapsed or the intercostal nerves were not carefully crushed or injected with alcohol at the time of the original procedure, general anesthesia may be more satisfactory, although the whole operation may be done under local infiltration if care and patience are exercised.

The former upper stage incision is reopened, making the incision curved if it was not formerly so, and the scapula mobilized by sharp dissection as far around as the origin of the serratus anterior muscle. The lateral edge of the erector spini group of muscles, which is no longer in its normal position but has dropped forward and inward with the collapse of the chest wall, must now be identified, freed from the regenerated rib, and retracted upward and medially. Care must be taken not to cut across its fibers transversely if scoliosis is to be avoided. In like manner, very forceful retraction of this muscle group in order to expose the transverse processes or a sweeping incision underneath the group in order to separate it from a series of ribs at one time, may likewise produce scoliosis by interrupting the nerve supply which arises from the intercostal nerves and extends posteriorly between the transverse processes.

Dissection is then directed against the regenerated ribs which will be found to run at a much more acute angle with the spine than the original ribs, freeing several of their periosteum posteriorly and laterally before attempting to section any of them. The reformed ribs will be found of irregular shape and contour, the size varying greatly according to age of patient, position, and the time since the original thoracoplasty. Not infrequently they will be found to join one another at various levels or even be fused into a very dense bony plate extending over several interspaces. Their thickness may vary from 2 or 3 mm. to 10 or 15 mm. or more. Through this regenerated bone, blood vessels and pieces of tissue may extend leaving fenestra or windows in the reformed rib. Anteriorly the regenerated ribs tend to become thin, flat, and even at times broader than the original rib with bone occupying the site previously bearing the costal cartilage Posteriorly at times, rib regeneration may be deficient, weak, or even totally absent if Zenker's solution or formaldehyde has been thoroughly applied to the periosteal bed in the region of the angle at the time of the original operation or if the periosteum has been inad-

vertently or intentionally removed. Old fractures and false joints are encountered frequently. There is usually a knoblike growth of regenerated rib in the region of the transverse process where the regenerated portion of rib may or may not have fused to the transverse process, depending upon whether the costotransverse joint surface was completely removed at the original operation.

The original rib between the transverse process and the body of the vertebra, if not disturbed at the previous operation, will be found of normal consistency. The costotransverse joint, if it still exists, is reopened and the rib excised inside of it toward the body of the vertebra for a distance of 2 or 2.5 cm. This may be done without disturbing the transverse process or its muscular attachments or the dorsal branches of the intercostal nerve supplying the erector spini muscles. Removal of the transverse process may be readily accomplished and has been done at times in this series, but we do not favor it as we feel that disturbing the remaining origins and insertions of the digitations of the erector spini group predisposes to a deforming scoliosis as well as invites injury to the dorsal branches of the intercostal nerves supplying the same group, producing the same effect. Resection of the regenerated ribs over five or six dorsal levels centering about the fifth or sixth will usually be necessary in order to mobilize adequately the chest wall overlying the uncollapsed cavity. The exact number and position, however, must be determined by the size and position of the unclosed excavation. All small splinters of bone which may have rigid attachment anteriorly or posteriorly must be carefully sought out and excised, or they may defeat the whole purpose of the operation. Great care must be exercised in removing the irregular regenerated ribs to avoid injury to the underlying pleura or lung if subsequent contamination of the operative field is to be prevented. The whole procedure is necessarily rather a tedious one, but, with all, rather well tolerated by the patients who are already accommodated to collapse. In seven instances, because of the patient's condition or the extensive nature of the reoperation required, it has been necessary to perform the reoperation in more than one stage. Removal of all regenerated rib from the anterior chest may not be necessary if the uncollapsed cavity is small and situated well posteriorly. If any of the posterior reformed rib must be left because of the patient's condition, the stumps of the first and second ribs, if not too long, will interfere less with collapse than those of any below, although the completed result may not appear so well on the x-ray films. The lateral and anterior portions of these ribs, however, should always be removed thoroughly, particularly if the original cavity has been apical and of fair size.

Following the removal of the regenerated ribs, some patients will present a very flexible chest wall which will collapse sufficiently of

itself or with the aid of external compression to produce the desired closure of cavity. If it seems reasonable, at the time of operation, to expect such a result, the incision may be closed in routine manner and the patient re-rayed, by the special technic previously described, to determine whether or not all cavitation is obliterated. Should these films demonstrate the presence of uncollapsed cavity, the incision should be reopened within two weeks if the patient's condition permits, before regeneration of rib and fixation of the chest wall occur and more regenerated rib removed or an extraperiosteal pack inserted or both. The rigid chest wall which does not collapse spontaneously to the desired position usually requires firm sustained pressure overlying the cavity to produce and maintain the desired collapse. This we have accomplished by the method to be described subsequently.

No attempt has been made save in one instance to perform an extrapleural pneumonolysis. This resulted in secondary perforation of the cavity wall by the pack. This patient is still alive and in good condition, with negative sputum, more than four and one-half years later but still presents a large chest wall defect and a bronchial fistula. Such a result should not be unexpected in the presence of large peripheral cavity and has impelled us to abandon the procedure. The same collapse can be obtained by the application of extraperiosteal pressure as by the use of an extrapleural pack without the danger of possible lung injury and contamination of the operative field during the extrapleural stripping. The pleura, subpleural tissues, periosteum, and intercostal muscles, vessels, and nerves lie between the extraperiosteal pack and the lung, in this method affording an adequate blood supply and protection against perforation of the pack into the lung itself. Following an extrapleural stripping, the pressure must be directed against the parietal pleura or even at times against the visceral pleura, offering a much greater chance for perforation or contamination.

Extraperiosteal pressure has been applied in three patients presenting a moderately flexible chest wall by detaching the serratus anterior muscle from its insertion into the vertebral border of the scapula, rolling it into a mass and suturing it under tension over the cavity by a series of forty-day chromic gut mattress sutures extending from the dense area of scar tissue anteriorly to the heavy fascia of the erector spini group, posteriorly in the region of the transverse process. Two of these patients obtained closure of cavity and cessation of all expectoration; while the third patient has a persistently negative sputum in spite of a small area which, from the x-ray films, we believe represents a small residual cavity. We have not used this procedure since 1933 in spite of the good results obtained because of its inherent disadvantages of affording a pack of limited size, of soft consistency,

and of a material which will subsequently atrophy, and because of the wing scapula which results from loss of function of the serratus anterior muscle.

For the past two years, in patients who require something more than reoperation alone to produce closure of cavity, we have favored the use of an extraperiosteal gauze pack because it affords controlled, firm, sustained pressure in the desired location for a sufficient period

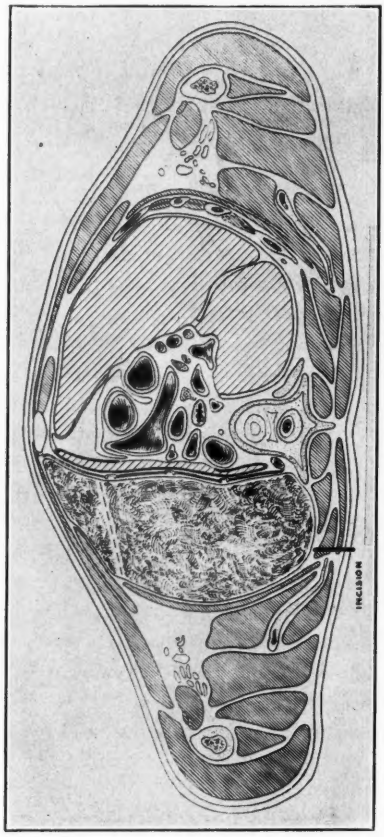

Fig. 1.—Topographic cross-section of chest following reoperation with extraperiosteal (subscapular) gauze pack.

of time to permit fixation of the chest wall in the collapsed position. This is applied in the operative field in the plane between the intercostal muscles and periosteum medially and the serratus anterior muscle and scapula laterally. (Fig. 1.) Fifteen to thirty square feet of gauze is tightly packed into the subscapular space depending upon the size of the subscapular pocket and the flexibility of the chest wall. A Penrose tube drain is inserted at the top and the bottom of the pack; the scapula is forced inward and the incision closed with inter-

rupted chromic gut sutures, approximating the cut edges of the rhomboid and trapezius above and the latissimus dorsi below. The packs in the first three patients, so treated, were saturated with 10 per cent argyrol. Because of the possible chance of argyria, its use has been abandoned and a 1:1,000 solution of neutral acriflavine in saline substituted. This has been found more satisfactory, as less secretion is produced in the wound.

X-ray films using the Bucky diaphragm according to the special technic previously described are made of the patient's chest a week or ten days following the insertion of the pack. Should there still be evidence of uncollapsed cavity, the wound is reopened, the pack removed, additional regenerated rib excised if necessary, and a larger pack inserted under greater pressure. If no cavity can be demonstrated, the pack is allowed to remain in place until the chest wall has become fixed in the collapsed position. The original packs were removed and replaced every ten days in the first two patients. All subsequent patients have been permitted to carry their packs unchanged for periods ranging from twenty-eight to forty-eight days, averaging thirty-six days. Removal of the original pack, resection of additional regenerated ribs, and reinsertion of a larger pack have been necessary in three patients. The period over which the pack is allowed to remain in place is determined somewhat by the flexibility of the chest wall at the time of reoperation, but should be sufficient to allow for fixation of this structure in the collapsed position.

Fixation of the chest wall in the collapsed position occurs either by deposition of scar tissue from the irritation from the gauze pack itself or by a regrowth of new bone in the periosteal bed or by a combination of the two. During the period when the patient is carrying the pack with the incision closed, there is considerable drainage around the tube, materially less with acriflavine than with argyrol. A small amount of 1:1,000 acriflavine is injected around each tube every two or three days in order to inhibit any secondary infection along this tract. The patient during this whole period of time runs a fever ranging from 101° to 102.5° F. per rectum. (Fig. 2.) Curiously enough the temperature gradually falls after the first two weeks, but in no instance has it reached normal during the period that the pack was in place. During this period of time the patient usually feels somewhat toxic and has an impaired appetite. Pain is usually less marked than following ordinary thoracoplasty. The leucocyte count has ranged from 6,000 to 15,000, the average being approximately 10,000 per c. mm., with an average polymorphonuclear count of 71 per cent. The hemoglobin in all of these patients has shown a distinct drop, averaging 32 per cent for the group. The sputum usually increases temporarily, then decreases, and becomes altered in character but may cease altogether. Some patients continue to raise mucoid

material and are troubled by a slight tickling cough during the whole period that the pack is in position. The urine shows a distinct yellow stain from acriflavine as long as the pack is in place, becoming less marked, however, after the first few days.

After the pack has remained in place for the desired length of time, the incision is reopened under local infiltration or gas anesthesia and

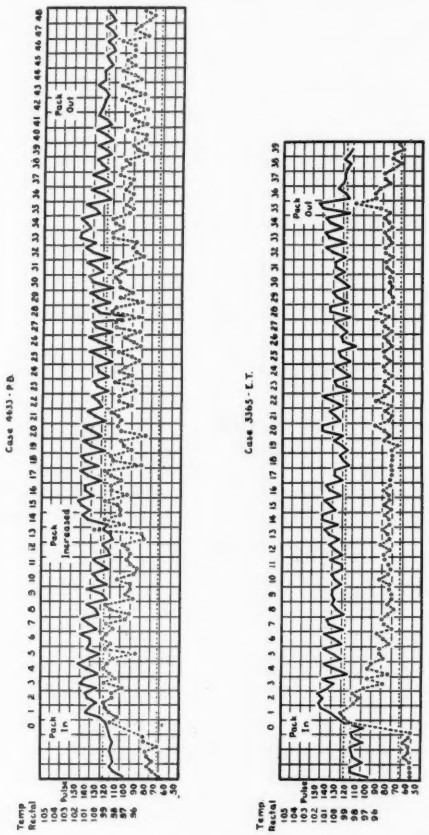

Fig. 2.—Temperature and pulse record during extraperiosteal pack.

the pack removed. A small amount of yellow-stained purulent material may be found about it, but the field is usually surprisingly clean. Smears from the exudate will usually show an occasional gram-positive coccus. The central portion of the pack will be found to have retained all its yellow stain; whereas the periphery where it is in contact with tissue will have lost some of the dye unless injections have been made repeatedly while the pack was in place. The gauze will be found somewhat adherent to the surrounding tissues in local areas, but may be readily separated. When the pack is removed, a varying number

Fig. 3. Fig. 4.

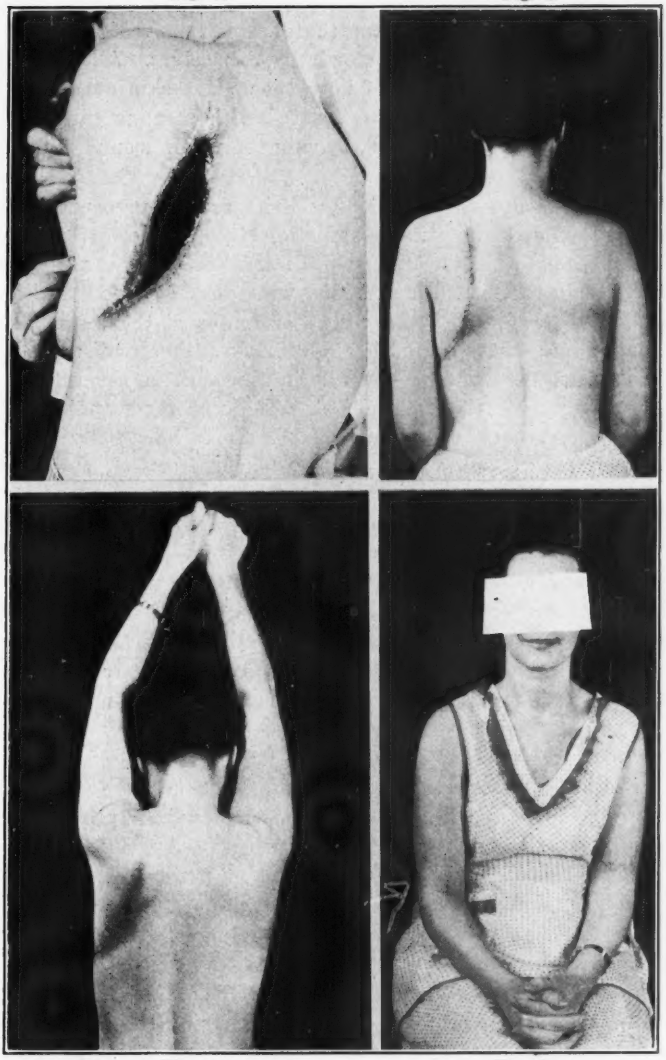

Fig. 5. Fig. 6.

Fig. 3.—Chest wall defect, six weeks following removal of gauze pack.
Fig. 4.—Back following plastic closure.
Fig. 5.—Arm function following plastic closure.
Fig. 6.—Anterior view dressed showing absence of deformity.

of yellowish stained devitalized tags of tissue firmly attached at one end may be found. These probably represent small tags of tissue with a poor blood supply which have become devitalized under pressure. They may be excised at the time the pack is removed, dissected off later or be allowed to separate slowly by themselves. All bleeding is controlled by the application of hot packs. The skin edges and exposed portions of the wound are covered with vaseline gauze, and a light pack of dry gauze inserted, leaving the whole wound wide open with no attempt at suture.

The wound is dressed daily under strict aseptic precautions until its depths are lined with clean granulation tissue. Firm external pressure is then applied anteriorly over the soft tissues of the chest wall approximating the lateral and medial walls of the wound, which are then allowed to adhere, obliterating the depths of the wound. This granulation and epithelization if allowed to continue undisturbed will eventually heal the wound completely in the course of several months, leaving a large epithelialized depression between the scapula and the midline. The process of complete healing may be greatly hastened and the cosmetic result vastly improved by performing a secondary plastic closure of the defect not obliterated by pressure, as soon as all slough has separated and the field becomes clean enough to permit this without too great danger of secondary infection. This has been performed under local anesthesia in from four to twelve weeks following the removal of the original tampon in all the more recent patients who have been treated by this method.

Following the removal of the pack, patients who have been somewhat toxic and febrile, with impaired appetite and moderate secondary anemia, begin to pick up almost immediately, losing their fever, regaining their appetite, and improving in weight and color. The irritating cough and mucoid expectoration resulting from presence of gauze in the wound usually cease almost immediately following its removal. After the plastic closure of such a defect, the scapula may be somewhat fixed for a short while, as it is sometimes following thoracoplasty, but it later loosens up and good shoulder function results. The cosmetic result obtained is almost identical with that following a radical upper thoracoplasty. (See Figs. 3-6.)

The clinical success of reoperation for inadequate thoracoplasty depends upon adequate removal of all rib overlying the area of uncollapsed cavitation and adjacent to it, plus, in suitable cases, the application of firm sustained pressure of sufficient degree to collapse the cavity and maintain it so until the chest wall has become fixed in this collapsed position. Forty patients (14.5 per cent of our thoracoplasty series) have been subjected to reoperative procedures of one type or another in an attempt to improve unsatisfactory thoracoplasty results. Twenty-three of these have been treated by reoperation alone and

seventeen by a combination of reoperation plus some form of internal compression; including one by extrapleural pneumonolysis, three by the use of the muscle pack, and thirteen by the use of subscapular gauze packs. Reoperation alone is the simplest procedure from the patient's standpoint and should be given first consideration. Its greatest use, however, will probably be found in those patients presenting smaller excavations, in whom a rather limited thoracoplasty of the Wilms-Sauerbruch or similar type has been performed previously. The performance of an adequate radical thoracoplasty of modern type in this group may offer a legitimate chance of obtaining the desired results if the chest wall is moderately flexible. If so, the use of subscapular packing is neither necessary nor desirable. The use of the serratus anterior muscle as a pack over an area of uncollapsed cavitation in patients with a flexible chest wall has given us two complete results, and one patient with apparent evidence of uncollapsed cavitation but a persistently negative sputum. We do not advocate its use, however, where direct pressure is needed badly, as it is quite likely to prove inadequate and the resultant wing scapula deformity is not altogether desirable. The use of extrapleural pneumonolysis following reoperation in the patient with a large peripheral cavity is not without considerable risk of cavity perforation or chest wall contamination and is not recommended.

If a cavity remains uncollapsed following a modern radical thoracoplasty and is accompanied by a positive sputum or other symptoms, the surgeon is confronted with a condition which requires more radical treatment than simple excision of the regenerated ribs. Some form of direct pressure over the cavity is necessary following reoperation if it is to be obliterated. The more rigid the pleura or underlying lung, or the larger the excavation present, the more necessary such direct pressure becomes. The application of an extraperiosteal (subscapular) pack of gauze, under such conditions, following the adequate removal of all regenerated rib in the particular region involved offers the most satisfactory method of converting the inadequate thoracoplasty into a satisfactory one. The procedure is not simple, either from the operator's or from the patient's standpoint. It has, however, been tolerated quite well considering the extensive nature of the surgery necessary and the condition of the patients on whom it must be applied.

RESULTS

The results obtained to date in this group of patients have been summarized in Table I.

From this total group, twenty-five of the forty have obtained closure of cavity, while thirty-one have obtained negative sputum. The use of some type of extraperiosteal pack has given us consistently better

results than reoperation alone, but it has likewise been used in the latter portion of the series where we have also had the benefit of experience with the previous portion of the group. It is quite probable that some of the unsatisfactory results recorded could have been improved had they been reoperated in the last few years. There have been two deaths following reoperation, both in 1930, but neither one associated with the use of any type of pack. One occurred twenty-six days, the other, thirty-one days postoperatively, but both from extension of tuberculosis. There have been five late deaths in this reop-

TABLE I

RESULTS OF SURGICAL REVISION OF UNSATISFACTORY THORACOPLASTY

Right—13		Left—27
Reoperation alone	Patients 23	Operations 30
Cavity		
Closed	12	
None originally	2	
Questionably closed	1	
Not closed	8	
Sputum		
Negative	14	
Negative before	1	
Positive	8	
Mortality		
Operative	2	26 days and 31 days
Late	5	7½ months to 4 years
Extrapleural pneumolysis	Patients 1	Operations 1
Cavity open	1	
Sputum negative	1	
Mortality	0	
Reoperation and muscle pack	Patients 3	Operations 3
Cavity		
Closed	2	
Questionably open	1	
Sputum negative	3	
Mortality	0	
Reoperation and extraperiosteal gauze pack	Patients 13	Operations 53
Cavity		
Closed	11	
Questionably open	2	
Sputum negative	13	
Mortality	0	
Total	Patients 40	Operations 87

erative series without the use of any packing, occurring from seven and one-half months to four years following the operation; one at seven and one-half months from tuberculous meningitis, one at ten months from dementia praecox, a third at one year from a progressive tuberculosis, the fourth at twenty months from pulmonary hemorrhage from an uncollapsed cavity, and the last, after four years from drowning. There has been no mortality in the series in which any form of extraperiosteal packing has been used; yet the results have been consistently much better.

CONCLUSIONS

Reoperation on patients whose thoracoplasties have proved unsatisfactory has given improved results in a considerable number. When combined with some form of extraperiosteal (subscapular) packing, the results have apparently been more certain, although the procedure is somewhat more formidable. The results to date justify a more extensive use of this procedure. The application of some type of subscapular pressure at the time of primary operation would seem advisable in certain selected patients and may obviate the necessity for reoperation later.

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RESECTION OF MEDIAL PORTION OF SCAPULA FOR RELIEF OF PAIN AND DISABILITY FOLLOWING THORACOPLASTY*

Report of a Case

C. R. Steinke, M.D., and J. T. Villani, M.D. (By Invitation)
Akron, Ohio

WHILE several operators have been removing the inferior border of the scapula to prevent it from overriding the remaining ribs in thoracoplastic procedures limited to the upper five or six ribs, the resection of the medial part of the scapula in connection with surgical treatment of pulmonary tuberculosis has not yet come to our attention.

CASE REPORT

E. L. H., white, American, thirty-two years old, was examined at the Akron Municipal Tuberculosis Clinic in August, 1931. There was evident retraction of the left chest wall anteriorly, and flat percussion note, bronchial breath sounds, and moist coarse râles were elicited over the left upper lobe. Roentgenographic study showed a large cavity 6×8 cm. in the left apex. Dense, soft shadows occupied the remainder of the upper half of the left lung. There was more discrete involvement of the lower lung. A small amount of infiltration was present in the right upper lobe. The sputum was positive for tubercle bacilli.

Artificial pneumothorax was instituted on the left with immediate symptomatic improvement. One month later the patient transferred to the Dayton, Ohio, Military Home. In January, 1932, the pneumothorax was discontinued, presumably because of adhesions and severe cardiac reaction. The ordinary hygienic regimen was then followed until June, 1933, when the patient removed to Tucson, Arizona.

X-ray examination at this time showed complete reexpansion of the lung, a slight diminution in the size of the left apical cavity, retraction of the diaphragm, and thickening of the pleura laterally. The lesions in the right lung had cleared (Fig. 1).

On July 15, 1933, a preliminary left phrenicectomy was done. Two days later the first stage of a left thoracoplasty was performed. The first four ribs were partially resected, and the periosteum was treated to inhibit regeneration. The transverse processes were not removed. Segments of the fifth to eleventh ribs were resected on August 4.

*Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery at New York, June 3, 4, 5, 1935.

Following the second stage, the patient had complete loss of function of left arm and shoulder. For three weeks he suffered severe to excruciating pain radiating from the scapula to the finger tips. The arm became markedly emaciated, and the upper arm and scapula were rigid. Through massage and passive exercise, 75 per cent of normal function was restored. Severe pain, however, caused by the scapula rubbing against the transverse processes continued. Purulent expectoration, positive for tubercle bacilli, persisted, although greatly diminished in amount.

On December 18, 1933, the patient underwent another operation. The scapula, which was found firmly embedded in scar tissue, was freed and elevated, and the transverse processes down to the ninth dorsal vertebra were removed, along with regenerated rib tissue.

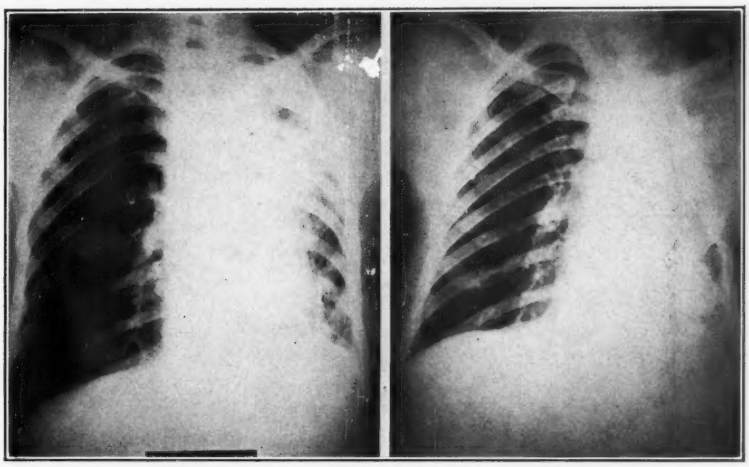

Fig. 1. Fig. 2.

Fig. 1.—X-ray of chest June 15, 1933, before surgical operations.
Fig. 2.—X-ray, April 23, 1934, after two-stage thoracoplasty and two accessory operations.

As only temporary relief was obtained, the patient was again operated upon February 26, 1934. The scapula was again found strongly adherent, and its lower border was wedged under the transverse processes. It was freed, the regenerated transverse processes of the seventh to ninth dorsal vertebrae were resected, and some rib stubs removed (Fig. 2).

The pain, however, did not disappear, and there was but little use of shoulder and arm. At a consultation held shortly after the fourth operation, Dr. C. A. Thomas of Tucson suggested the removal of a portion of the scapula, but this was thought inadvisable by the other consultants.

The patient later moved to Ann Arbor, Michigan. There two supplementary operations by Dr. John Alexander on August 13 and September 18, 1934, effected a complete closure of the cavity and the disappearance of tubercle bacilli from the sputum. In these procedures all regenerated rib tissue and transverse processes and the remaining anterior segments of the upper four ribs with their cartilages were removed. The scapula was again freed from the surrounding scar tissue (Fig. 3, left).

On October 23, 1934, the patient was admitted to The Edwin Shaw Sanatorium, Akron, Ohio. Besides the emaciation and weakness resulting from the recent operations, the presenting symptoms were fixation of left scapula, severe pain caused by friction of the medial

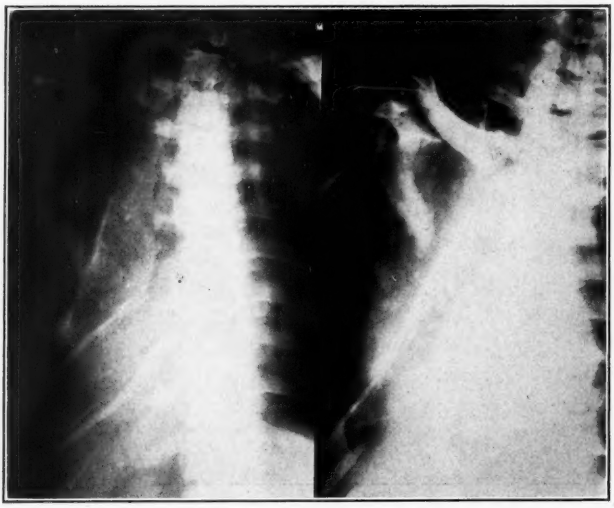

Fig. 3.—Scapula before and after resection of medial portion.

border of the scapula against the vertebral bodies, and marked limitation of motion of the left arm. The arm could not be raised above the level of the shoulder, nor carried back of the 180° angle with the body. Forward motion was limited to thirty degrees forward from the 180° angle.

For several months conservative measures were employed. These included massage, active and passive motion, infra-red rays, and counterirritants. There was no abatement of pain or improvement of function. Daily doses of codeine and analgesics were necessary.

Resection of the medial portion of the scapula was finally decided upon and was performed February 28, 1935, at the Akron City Hospital. Nitrous oxide oxygen anesthesia was used. The incision was parallel and slightly lateral to the old thoracoplasty incision. The

scapula was found displaced medially and anteriorly, its inner border extending along the left border of the vertebral bodies. Very dense fibrous tissue held the under surface. The scapula could not be elevated until this scar tissue was severed. The suprapinatus and infraspinatus muscles were stripped back from their origin in the scapular fossae to the line of resection. The teres major was also sectioned at its origin. The medial segment of the scapula, 6 cm. wide at its upper border and 9 cm. at the lower portion, was then resected. Around the cut edge were sutured the free borders of the supra- and infraspinatus muscles. The regenerated transverse processes of the eighth and ninth dorsal vertebrae and some regenerated rib bodies were then removed. A cigaret drain was placed in the lower angle of the incision. Two rows of chromic sutures were used to approximate the muscles and fascia, and dermal sutures for skin closure (Fig. 3, right).

To prevent immobilization of the scapula, active and passive motion were instituted as soon as the incision was healed. Furthermore, the left arm was allowed to hang over the edge of the bed several hours a day while the patient lay in the prone position.

RESULTS

There has been complete restoration of mobility to the left arm. It can be elevated and rotated through the normal range without pain or hindrance, except as due to the residual muscular atrophy. There is no impingement of the scapula on ribs or spine.

The severe pain caused by the rubbing of the scapula against the vertebral bodies and transverse processes has disappeared. There is present, however, moderate myalgic pain along the resected edge of the scapula.

DISCUSSION OF PAPERS ON "BRONCHOSCOPY FOLLOWING THORACOPLASTY FOR TUBERCULOSIS," BY CABITT, SINGER, AND GRAHAM; "SURGICAL REVISION OF UNSATISFACTORY THORACOPLASTY BY REOPERATION AND EXTRAPERIOSTEAL PACKING," BY KINSELLA; AND "RESECTION OF MEDIAL PORTION OF SCAPULA FOR RELIEF OF PAIN AND DISABILITY FOLLOWING THORACOPLASTY," BY STEINKE AND VILLANI

DR. POL. N. CORYLLOS.—Dr. Graham in his paper told us that in judging the results obtained with thoracoplasty in pulmonary tuberculosis we should not insist only upon the mortality of this therapeutic method but also, and even more upon the number of operated patients who were well five years after operation. These are courageous and wise words. I perfectly agree with Dr. Graham that if we operate only upon cases with cavities and not upon chronic proliferative cases—which remain well without thoracoplasty—we are bound to have operative deaths or deaths from further extension of the disease. But if only 50 per cent of our operated cases are well after five years, we can say that we have saved at least 40 per cent of cases doomed to certain death. And when we operate upon cases with cavities in

both lungs, even if our mortality is 60 per cent, we have saved 40 per cent of condemned cases. With Dr. Graham, I believe that these results justify our present efforts; furthermore it is not by avoiding the issues but by persisting in our efforts that we shall perfect our technic and better define the indications; increased experience and knowledge will permit us to continue to improve our results and at the same time tackle with increasing success the more advanced cases. I had such an unexpected result lately, on a young girl with multiple large cavities of the right lung, who had been bleeding for twelve days. Pneumothorax was unsuccessful and phrenic exeresis did not stop hemoptysis. I performed a three-rib upper thoracoplasty; the patient stood the operation surprisingly well and bleeding stopped.

On the other hand, I am not certain that lipiodol injections in tuberculous patients are harmless. I had a number of very serious reactions, especially in cases in which lipiodol was injected into the cavity through the chest wall according to the procedure of Ameuille. A patient in whose bronchi a few cubic centimeters of skiodan were injected developed an acute tuberculous bronchopneumonia and died within twelve days.

Dr. Kinsella advises the combination of gauze packing and thoracoplasty in order to produce additional compression upon obstinate cavities, as was already proposed by Bernou in France and by Casper in this country. I published some time ago my results with this procedure. They were not satisfactory, and I have abandoned this method. There are huge cavities which are collapsed with thoracoplasty. There are others which do not collapse whatever you may do; resection of transverse apophysis, anterolateral, extended or improved thoracoplasties, and gauze or paste packing. Autopsies of such cases have shown us that in shelled-out upper lobes in which the cavities open directly into a large bronchus, closure of the cavity is an anatomic and physiologic impossibility. I shall show a few slides illustrating these points. I shall further develop this point in my paper on the management of bilateral cases.

Incidentally, I want to insist again upon the inaccuracy of the term "compression of the lung" following thoracoplasty. We do not and we cannot compress the lung by this procedure, the aim of which is to deprive the chest wall of its rigidity and change it to a soft wall akin to the abdominal wall. Under such conditions the lung will collapse because of the collapse of the chest wall. But under no circumstances can it be compressed because under no conditions can the pressure exerted upon the chest wall exceed the intrapulmonary pressure; it is obvious that both are equal to atmospheric pressure.

I agree with Dr. Steinke on the usefulness of the resection of the posterior border of the scapula. I have repeatedly performed this simple and harmless operation, especially for mobilizing anchored scapulae. In one case I was obliged to resect four-fifths of the blade of the scapula and use the subscapular and supra- and infraspinatus muscles as a flap for closing a huge cavity which remained after a Casper operation. The results, both anatomic and physiologic, were excellent.

DR. E. S. WELLES.—I wonder whether the Society as a whole always appreciates Dr. Graham. Time after time we have come to these meetings, and either in the papers which Dr. Graham has presented or in discussion which he has offered following other papers, sometimes favorable discussion and sometimes unfavorable comments on what has been presented, Dr. Graham has given us again and again evidence of what we might call wisdom, which is a rare faculty in the medical profession. I greatly appreciate his very short talk today on thoracoplasty statistics and the fact of getting down to the meat of the problem of what we are doing and

why, regardless of our percentages of mortality and so on. The problem is what to do for the patient. If we lose some patients trying to do it, we are working in a good cause.

Recently I have operated on quite a number of patients for thoracoplasty, and if I presented them today, my presentation would sound a little bit like that which Dr. Torek gave us yesterday, of three or four trials and a hundred per cent mortality so far. That is very bad for my statistics on thoracoplasty, but, on the other hand, I am working hard and trying to retrieve some of the lost causes, and I hope at the next meeting to offer a paper to the Program Committee which will be a discussion of operative procedures on bad risks.

You remember a good many times in the past years the subject of Dr. Archibald's famous description of the thoracoplasty patient has come up, "the good chronic," and naturally if we operate on Dr. Archibald's good chronic, we are going to have good results. In recent years thoracoplasty has been extended by others to patients who are hardly yet in the good chronic stage, I mean they have not been that sick, and statistics are getting better and better. On the other hand, I do not believe that we ought to pay attention to statistics, and a group of us in Saranac Lake have been trying to figure out something to do for the very bad chronics, the bad risks.

Dr. Coryllos and Dr. Ornstein presented a paper yesterday on very bad cases. All these bilateral cases on which they were operating are as far removed from the original indication of thoracoplasty, the good chronic, as could possibly be, and that is the type of case I am talking about, not necessarily the bilateral one, although some of them are, but others are unilateral cases, although they are not fit to operate on. On the other hand, they are going to die, obviously, if something is not done, and I have been trying to do some operations on them. I have not had much luck so far, although I have had some. Any patient that we pull through is clear profit, because the rest are all total losses.

I say again that I appreciate Dr. Graham's bringing up to us again so wisely this talk about statistics.

Dr. Kinsella's paper about reoperation and removal of these regrown ribs is very interesting. I think the one point ought to be brought up that it certainly is a dangerous operation whenever it is done. I am not offering that as an argument against it, but it ought to be emphasized that the removal of these horrible looking pieces of regrown rib that he showed on the slide is extremely difficult, and there is always danger of tearing into the cavity, making a hole right into the lung, which makes an unpleasant complication, and the whole operation is a very much more severe one, harder on the patient, than the original thoracoplasty.

Dr. Coryllos has started at us again on our nomenclature in his talk about the word "compression." I have been a little surprised that he did not compliment the speaker who used the term "pneumonolysis" this year. We have had many papers on pneumolysis in this Society. The subject of pneumonectomy, and so on, come up. Dr. Coryllos has got us so well educated now that he does not have to mention the fact that we say pneumonolysis instead of pneumolysis, and we are now freeing the lung from its adhesions instead of freeing air, as he might have brought up the other time.

DR. HOWARD LILIENTHAL.—Permit me to call attention to the importance of bronchiectasis as a producer of sputum in patients who have been operated upon for tuberculosis by pulmonary compression. I said "compression" advisedly. I do not mean collapse, I mean compression, and yet, in spite of the compression, there may be bronchiectasis from which the sputum comes. It seems to me that something ought to be done for these people, because they cannot get away from the idea that they are tuberculous, and their friends certainly do not get away from that idea.

It strikes me that it would be a good idea—I have tried it in one case with only moderate success, but the patient is still under observation—to employ the method of Berck, with the carcinoma dose of x-ray in order to stop the secretion from the dilated bronchi, or to minimize it at any rate.

Now about repeated and multiple operation. I do not hear so much about re-operations for the removal of more rib by going through the axilla or anteriorly. I would call attention to the fact that it is not necessary to operate by a second incision in the axilla. By incising through the original scar there is very little bleeding, and by retracting the scapula further forward, which is much easier after the original thoracoplasty has been performed, you will be able to resect the anterior stump as far as the sternum, should this seem desirable bleeding. I had one patient who had to be reoperated upon, and I removed ten ribs at one sitting with the so-called radio knife and without the ligation of a single vessel. Primary union followed.

Another point—avoid gauze packings for purposes of compression. Use crumpled rubber dam, not rubber tissue, and a thin layer of gauze to cover it. From three to six, or even eight days later, according to the patient's condition, remove the rubber dam where it has protruded from the lower end of this wound and you will find that the cavity is three or four times as capacious as it was when you put the rubber dam in. The space may then be drained by tube. Granulation tissue will form, and the collapse will progress. The method is especially valuable in apicolitic thoracoplasty, in which four or five of the upper ribs are resected.

Rather than to cut the scapula I prefer the resection of another rib.

DR. R. B. BETTMAN.—I wish to discuss briefly three points. For years I have performed an apicolysis as part of the technic of an upper thoracoplasty in those patients in whom a large apical cavity exists. Toward the end of the operation, after the upper four, five, or six ribs, depending upon the case, have been resected, the finger is inserted under the intercostal muscles in one of the interspaces close to the spine, and the lysis is accomplished by careful, gentle, digital dissection. The entire apex can be easily collapsed. A pack will not be necessary. It goes without saying, of course, that the entire first and second ribs have been removed and practically the entire third rib. I have found that, in this manner, I have been able to close large cavities which otherwise would have remained open.

I think that there is a very definite place for partial thoracoplasty in the treatment of localized tuberculosis. We have all seen cavities close after the so-called selective pneumothorax, and the partial thoracoplasty works the same way. I agree with Dr. Kinsella, however, that this type of operation has certain dangers. It can be done only on the most carefully selected patients, and, in case of doubt, it is unquestionably safer to err on the side of removing more ribs than too few.

Lastly, I want to thank Dr. Steinke for bringing to discussion the problem of the fixed scapula. I agree with Dr. Lilienthal that, as a rule, most of the trouble from the scapula occurs in patients in whom, following a partial upper thoracoplasty, the scapula slips under the remaining sixth or seventh rib. In such instances I have followed the procedure mentioned by Dr. Lilienthal, namely, removal of the rib rather than the removal of part of the scapula. Occasionally I have seen trouble from the scapula fixed in the same manner that Dr. Steinke discusses, and heretofore I have tried to remedy the condition by freeing the adhesions and attempting to elevate the scapula by pulling the long muscles of the back into the defect left after the ribs have been removed. I think, however, that removal of the portion of the scapula, as indicated by Dr. Steinke, would be not only simpler but more beneficial.

DR. DUFF S. ALLEN.—There is one point I should like to discuss in regard to the paper of Dr. Kinsella. This concerns the removal of the newly regenerated portion of ribs. For several years it has been our routine to remove the regenerated portions of the ribs involved in the previous stage of a multiple stage thoracoplasty along with additional segments of the same ribs.

Our reasons for doing this were based on the fundamental conception that whenever a patient dies immediately following thoracoplasty more often than not the death can be attributed to the removal of too *much* rib. This does not necessarily mean too *many* ribs, since it may result from the removal of too long segments of a few ribs. Each patient presents an individual problem. In patients who are poor surgical risks or in the very far advanced cases such double resections of the ribs are especially indicated.

At first, like Dr. Welles, we were concerned about the dangers of entering into the pleura or into cavities in the lung during the resection of the regenerated portions of the ribs. I am pleased to say we have not had a single unfortunate experience of this sort in more than one hundred operations carried out in this manner. There are a few valuable points which may be observed in removing these regenerated ribs. First, the normal unresected rib is exposed distal to its previously resected and regenerated portion. This unresected normal rib is then cut across and lifted upward toward the ceiling with a strong instrument. The regenerated portion can now be dissected free from the parietal pleura with sharp dissection and under direct vision. The dissection is begun at the distal end of the regenerated rib and is carried backward toward the spine.

Removal of regenerated portions of ribs along with additional lengths of the same ribs is not attended by a great deal of shock. It has seemed to us, in fact, that there is less shock following removal of a long segment of a rib by this two-stage procedure than following removal of a similar long segment at one sitting.

DR. JOHN ALEXANDER.—Although our experience with secondary thoracoplasty has been relatively small, it has indicated that the operation is highly efficient in the closure of residual cavities; therefore the obvious disadvantages of gauze packing can usually be avoided. We have undertaken secondary thoracoplasty in 14 patients, one of whom was found to have such a thick, dense, and continuous wall of new bone that it could not be safely removed. The cavities were closed, and the sputum became consistently negative in 12 of the remaining 13 cases in which the operation was actually performed.

We have routinely resected the entire length of the transverse processes of the vertebrae and necks of the ribs at, above, and below the level of the pulmonary cavity in both our primary and secondary thoracoplasties during the last two or three years. I believe that this is an important improvement in the technic of thoracoplasty and that it has increased the closure of cavities by from 10 to 15 per cent. We had feared that the removal of the transverse processes might produce troublesome scoliosis, but there seems to be even less scoliosis than before; the explanation for this unexpected finding is probably that we have, during the last few years, routinely used special postoperative measures to prevent scoliosis.

DR. EVARTS A. GRAHAM.—I just want to say a word. I made a statement in my remarks that I had not seen any extensive series of cases of thoracoplasty reported which had been studied by bronchography. Somehow or other I forgot momentarily the important paper of Dr. Neuhof which was published last year in the *American Journal of Roentgenography*, which is a study based on some sixty or seventy cases of thoracoplasty with bronchography. His paper is mentioned in our manuscript.

My only plea was that we should use the procedure more extensively than it has been used.

DR. THOMAS J. KINSELLA.—I appreciate this discussion very much. It has been our practice in a number of these patients to reoperate and take out the regenerated rib as well as any unresected at the original operation, then close the incision, wait ten days or so and make special x-ray films of the collapsed lung. If there is still evidence of uncollapsed cavity, the incision is reopened and a gauze pack inserted under pressure.

I appreciate Dr. Lilienthal's suggestion for the use of rubber dam in place of gauze for this procedure. I wonder, however, whether five days is a sufficiently long period of time to maintain pressure on the chest wall in order to be certain that it remains in the collapsed position to insure closure of some of these cavities. I do not like to use gauze unless I have to, but it has helped us to close some cavities which we have been unable to obliterate by any other method.

The matter of perforation of the cavity at the time of reoperation is something to be considered. Removal of irregular regenerated ribs certainly is accompanied by some possible chance of injury to the pleura. If the dissection is done carefully and gently, this danger may be held to a minimum.

I do not believe that apicolysis is without some danger of perforation of the lateral cavity wall, particularly in very large peripheral cavities. For this reason, we have not adopted the procedure as either a primary or a secondary operation.

DR. CARL R. STEINKE.—In answer to Dr. Lilienthal and Dr. Bettman's suggestion that more ribs be taken out, I think you noticed in the last x-ray lantern slide, that when Dr. Alexander got through operating there were no more ribs left to be taken out, at least down to the ninth rib. Compression therapy was used as an aid in collapsing the cavity, a thick pad being held firmly against the chest by straps to force in the chest wall further. This pressed the scapula in against the edge of the vertebrae for a long period of time, allowing it to become very firmly adherent. The scapula was fixed and held in this position. This type of compression probably is a splendid adjunct in treatment and a good thing to aid in collapsing the cavity. One of the important things in after treatment, following the freeing and resection of the scapula, was allowing it to hang over the edge of the bed. This man, previously, had had the scapula freed three times, making the scar more dense than it would have been originally.

A ROENTGEN STUDY OF THE MODE OF DEVELOPMENT OF ENCAPSULATED INTERLOBAR EFFUSIONS*

LEO G. RIGLER, M.D.
MINNEAPOLIS, MINN.

THE wealth of literature concerning interlobar collections of fluid attests the general interest in this subject. In spite of this, the exact pathogenesis of this condition is not entirely clear, and two possible mechanisms by which fluid may become encysted in the interlobar pleural space have been considered.

The first type of development of encapsulated interlobar effusions has been strongly emphasized by Lilienthal,[3] Sauerbruch,[7] and others; it is considered by most observers to be the usual method by which this process occurs. This theory presupposes that the accumulation of fluid in the interlobar space is the result of an infection of this portion of the pleural cavity itself, independent of, or simultaneous with, an infection of the remainder of the pleural cavity. The infection may be the result of the extension into the space of bacteria from the contiguous lung, such as occurs in pneumonia or tuberculosis. Rupture of a small lung abscess through the visceral pleura lining the interlobar fissure is another typical example.

This concept of the process considers the infection of the interlobar fissure as the primary pleural lesion. Lilienthal,[3] in fact, describes a gradual increase in the amount of fluid in the interlobar space until rupture or extension into the general pleural cavity occurs. The invasion of the peripheral pleura is thus secondary to the involvement of the interlobar pleura.

Stewart,[9] Austrian,[1] and others have ascribed the occurrence of encapsulated interlobar effusions in cases of cardiac failure to a previous obliteration of the peripheral pleural cavity leaving the interlobar spaces as the only ones into which transudation could occur. Here again the accumulation of fluid between the lobes is looked upon as the primary pleural process. More recently Stein and Schwedel[8] have reported that encysted interlobar effusions in patients with cardiac failure are not uncommon. In their cases the simultaneous presence of fluid in the peripheral pleural cavity and in the interlobar pleural spaces seemed to preclude the explanation given above.

*From the Department of Radiology of the University of Minnesota and the University Hospital.

Read at the Eighteenth Annual Meeting of the American Association for Thoracic Surgery in New York, June 3, 4, 5, 1935.

There is another hypothesis which assumes that an interlobar collection of fluid is the residue of a general pleural effusion. This has been but little emphasized; although there are reports of cases showing, e.g., as Wessler and Jaches[11] do, simultaneous free pleural effusions and encapsulated interlobar collections. Stoloff[10] also cites such cases in children, but there is no comment as to the mechanism by which this occurs. Fleischner,[2] in his admirable monograph on interlobar pleurisy, calls attention to the extension of a general empyema, particularly in children, into the minor interlobar fissure but does not discuss it further.

Some years ago, while engaged in a study of the movement of pleural effusions with change in position of the patient, I observed a process concerning which little or no reference in the literature could be found. It was reported at that time,[5, 6] that fluid in the interlobar

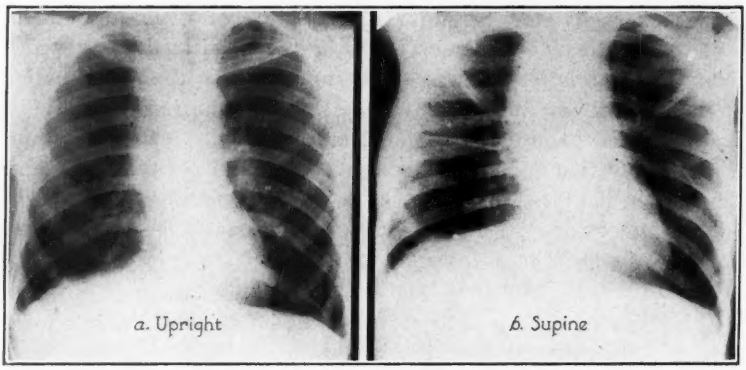

Fig. 1.—Case of Hodgkin's disease with small pleural effusion right side.
a, Posteroanterior roentgenogram, upright position, showing slight obliteration of costophrenic angle but no shadow of minor interlobar fissure.
b, Anteroposterior roentgenogram, supine position, showing broad, dense, horizontal band in region of minor fissure, obviously due to extension of free effusion into the interlobar space.

fissure could be demonstrated by roentgenograms made with the patient in one of the horizontal positions, particularly lying on the affected side; this fluid was apparently not present in the films made with the patient upright. This finding is well demonstrated in some of the cases reported in those two papers and is further shown in Fig. 1. This represents a case of Hodgkin's disease of the mediastinal lymph nodes. It will be noted in Fig. 1*a*, a posteroanterior roentgenogram of the chest made in the usual upright position, that no shadow of the minor interlobar fissure on the right side can be made out. There is a slight obliteration of the costophrenic angle on this side suggesting either a small amount of pleural fluid or pleural adhesions. In Fig. 1*b*, an anteroposterior roentgenogram made at the same time

with the patient supine, a broad dense horizontal band crossing the right lung in the position of the minor interlobar fissure can be made out. This patient gradually developed sufficient fluid in the right pleural cavity to be unmistakable. The conclusion that the change in appearance of the roentgenogram from upright to supine positions was due to extension of a small general pleural effusion into the interlobar pleural space seems reasonable.

Another example of this phenomenon is shown in Fig. 2. This was a case of cardiac failure with a bilateral pleural transudate of small amount. In Fig. 2*a*, a posteroanterior roentgenogram made with the patient upright, the fluid in the right pleural cavity can be made out. There is also shown a fine line in the region of the minor interlobar fissure. With the patient in the right lateral decubitus position,[5] at the same sitting, a posteroanterior roentgenogram was made and is

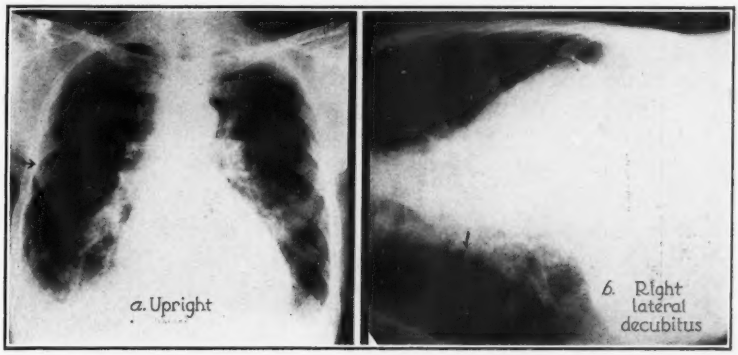

Fig. 2.—Case of cardiac decompensation with small bilateral pleural effusion.

a, Posteroanterior roentgenogram, upright position, showing fluid in both costophrenic angles. There is a fine linear shadow (arrow) in the region of the minor interlobar fissure.

b, Posteroanterior roentgenogram, right lateral decubitus position. Note the marked increase in size of the interlobar fissure shadow (arrows) due to the extension of fluid into this space.

shown in Fig. 2*b*. Here the minor interlobar fissure shadow has broadened out a great deal simulating an encapsulated effusion in the interlobar space. There is obviously a striking difference in the appearance of the pleural shadow in the two positions.

While these two cases are illustrative of numerous others with pleural transudates, the same process may be observed in exudates, both purulent and nonpurulent. An example of this type is shown in Fig. 3, a case of tuberculous pleurisy with a characteristic serofibrinous exudate in the right pleural cavity. In the upright posteroanterior roentgenogram (Fig. 3*a*) there is a slight suggestion of a pleural effusion on the right side but it is doubtful. There is no shadow whatever in the region of the minor interlobar fissure. In

the lateral decubitus position, however, the posteroanterior roentgenogram (Fig. 3*b*) shows the typical appearance of a moderate amount of fluid in the inferior costal gutter—the usual finding when this position is used. Furthermore, in this position, the fluid may be seen extending into the minor interlobar space producing a somewhat triangular-shaped shadow with its base at the periphery. These observations indicate clearly that, under certain conditions, fluid, often so small in amount that it is not readily detected, may extend from the general pleural cavity into the interlobar spaces.

Because of the anatomic character of the minor interlobar fissure on the right side, abnormalities in this space are much more readily detected roentgenologically than are corresponding changes in the major fissures. It is for this reason that only the minor fissure has been considered in this discussion. It should be pointed out that the same processes which affect the minor fissure will no doubt affect the major

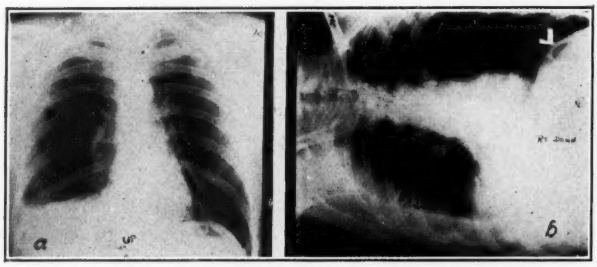

Fig. 3.—Case of right-sided tuberculous pleurisy with small effusion.

a, Posteroanterior roentgenogram, upright position, showing flattening of diaphragm and obliteration of costophrenic angle but no shadow in region of minor interlobar fissure.

b, Posteroanterior roentgenogram, right lateral decubitus position, showing considerable fluid (arrow) in inferior costal gutter. Note the shadow of the minor interlobar fissure (arrow) into which fluid has extended with the change in position.

ones—in fact, changes exactly similar to those reported here have been observed in the major fissure. For the sake of clarity and brevity of description, however, the changes occurring in the minor fissure alone will be detailed.

Observation of an extensive series of cases of pneumonia with serial, often daily, roentgenograms, made at the bedside, with the patient supine, reveals another phenomenon which may be directly correlated with the above data. Frequently during the course of a right lobar pneumonia, often within twenty-four hours after the onset, there appears in the roentgenogram a fairly broad, dense, linear shadow, extending horizontally across the lung and corresponding exactly to the position of the minor interlobar fissure. This shadow most commonly disappears after the pneumonia has fully resolved. A typical

case illustrating this point is shown in Fig. 4. This patient developed lobar pneumonia twenty-four hours prior to the first roentgenogram. In this was shown the consolidation at the right base together with a very faint horizontal linear shadow in the region of the minor interlobar fissure. Two days later (Fig. 4*a*) the shadow of the fissure appears much wider and simulates encapsulated exudate. The diagnosis of free effusion was made at this time because of this shadow, and two days later 250 c.c. of serous, sterile fluid was removed by paracentesis from the pleural cavity. Ten days later (Fig. 4*b*) the shadow of the interlobar pleura had almost completely disappeared. This absorption occurs very commonly even though aspiration is not done.

In the previous literature this shadow has been deemed to be due to thickening of the pleura, but its rapid appearance and complete

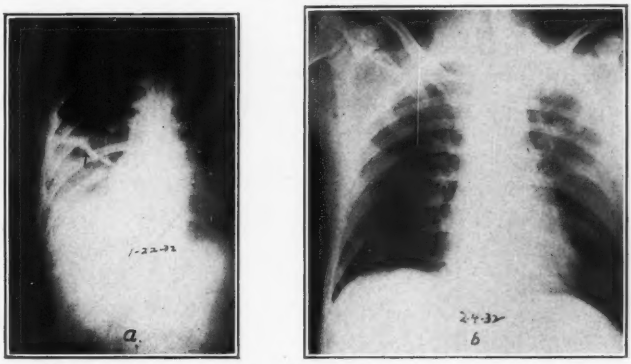

Fig. 4.—Case of lobar pneumonia right middle and lower lobes.

a, Anteroposterior roentgenogram made January 22, 1932, two days after onset. Note the broad, dense, horizontal shadow (arrow) characteristic of fluid in the minor interlobar fissure.

b, Same case twelve days later showing resolution of pneumonia with almost complete disappearance of the interlobar fissure shadow (arrow).

disappearance make this seem highly improbable. The best explanation for this occurrence, particularly when correlated with the observations recorded above, is afforded by the fact that many cases of pneumonia are accompanied by small amounts of pleural fluid either serous or purulent. According to Lord,[4] Maragliano found fluid in the pleural cavity in 38 of 58 cases of pneumonia; Sello demonstrated fluid in 2 cases at the very beginning and in 31 during the course of the disease. Postmortem studies serve to substantiate this high incidence, effusion of some type being found in about one-third of the cases.

From these facts it appears likely that this shadow of the interlobar fissure, appearing during the course of lobar pneumonia, is due to an extension of a small general pleural effusion into the interlobar

space, the patient being in the supine position. The opportunity to observe this process, together with the development of the encapsulation which may result from it, was afforded in the following case which is reported in detail.

CASE REPORT

B. P. a white male, aged twenty-two years, became ill on Dec. 12, 1932 with chills, vomiting and fever, the temperature rising to 104°F. He was admitted to the Students' Health Service in the University Hospital on December 14, at which time his temperature ranged from 103° to 104.5°F. His leucocyte count was 28,000 and his sputum showed pneumococci of Group IV. There were few physical findings other than a pleural friction rub at the right base.

A roentgenogram made on December 15, at the bedside with the patient supine, is shown in Fig. 5*a*. This gives evidence of consolidation at the right base and ex-

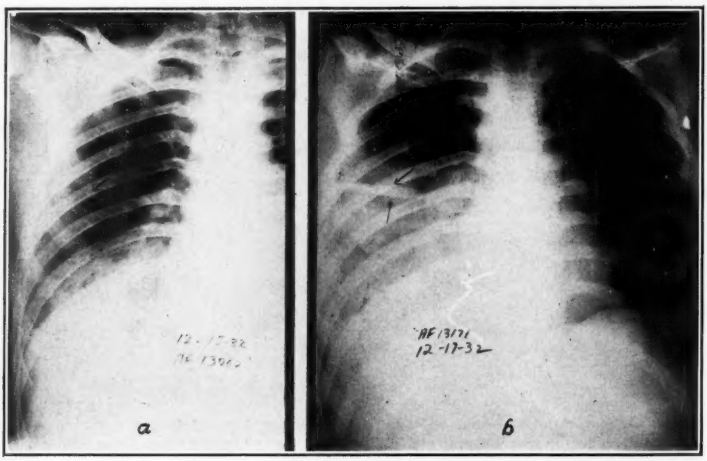

Fig. 5.—Case of lobar pneumonia developing encapsulated effusion in minor interlobar fissure.

a, Anteroposterior roentgenogram made December 15, 1932, three days after the onset. Note the consolidation at the right base and the fine linear shadow (arrow) of the minor interlobar fissure.

b, Same case two days later showing increase in consolidation, fluid in the free pleural cavity, and a marked broadening of the minor interlobar fissure shadow (arrow) which is now a horizontal band.

tending upward. A distinct horizontal linear shadow in the region of the minor interlobar fissure may be seen. On December 17 a second film (Fig. 5*b*) showed an increase of the consolidation with a diffusion of the shadow. In addition the linear interlobar fissure shadow had widened markedly, representing a broad band rather than a line. From this it was concluded that there was free fluid in the pleural cavity and the following day 250 c.c. of serous fluid was aspirated. This showed Group IV pneumococci on culture. On December 21, 500 c.c. of slightly turbid fluid was aspirated. At this time another roentgenogram showed a striking change in the interlobar fissure shadow. This had increased in size and assumed an oval shape characteristic of an encapsulated interlobar effusion. The findings were very similar to those shown in Fig. 6*a*. The temperature still remained very high. Intercostal closed drainage of the general pleural cavity was instituted, but no effort was made

to drain the interlobar space. After a few days the fluid in the peripheral pleural cavity diminished, and there was little or no further drainage. On December 27 the roentgenograms shown in Fig. 6 were obtained, localizing clearly the encapsulation in the minor interlobar fissure. At this time there was no further evidence of fluid in the main pleural cavity, and the consolidation had been resolved almost completely. Aspiration of the minor fissure produced 100 c.c. of pus and serous material in which pneumococci were found. A rib resection was done on December 30; the interlobar space was entered and a cavity about 9x7 cm. was found. Air was injected after removal of fluid and a Penrose drain inserted. Further roentgenograms with air in the pleural pocket demonstrated the exact limits of the space. Reaccumulation of fluid within the space could be readily demonstrated although it was now well separated off from the general pleural cavity. Eventually improvement gradually occurred, and the patient was discharged with complete recovery on March 2, 1933.

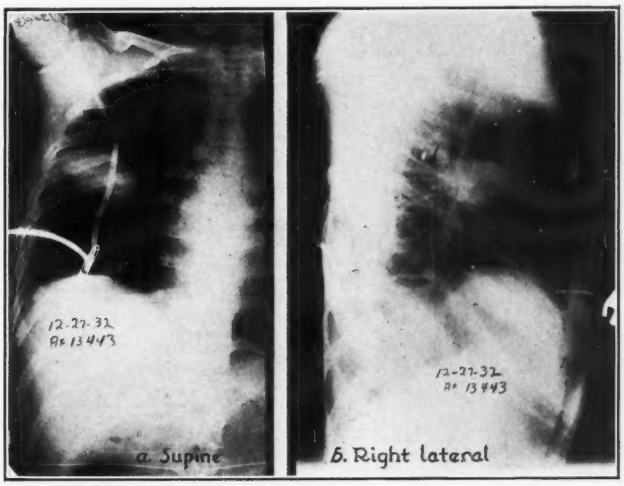

Fig. 6.—Same case as in Fig. 5.

a, Anteroposterior roentgenogram made December 27, 1932. The drainage tube is in the general pleural cavity. Note the characteristic oval shadow of an interlobar encapsulation. This was already clearly present six days earlier. Note the absence of fluid in the free pleural cavity at this time.

b, Lateral roentgenogram made at the same time. This shows the posterior position of the drainage tube and the typical anterior position of the encapsulated effusion in the minor interlobar fissure.

COMMENT

The case reported above seems to consolidate the observations on the other cases and to indicate a type of mechanism for the formation of an interlobar encapsulation which may best be summarized by reference to the drawing shown in Fig. 7. This is a diagrammatic representation of the roentgenographic findings in a large number of cases. The pleura itself is shown in black, the potential pleural cavity is white and has been enlarged for purposes of clarity. Pleural fluid is shown by the stippling, while pleural adhesions are represented by short, solid, black lines. In the first stage of a pleural effusion, with

only a small amount of fluid and with the patient upright, the fluid accumulates largely below the dome of the diaphragm extending upward around the periphery because of the retraction of the lung. It does not rise enough to reach the minor fissure; hence no shadow of this fissure is seen. In the prone or supine position, the fluid extends in a thin layer to a higher point, reaching the interlobar fissure into which it is drawn by the capillary pressure of this small space. In the lateral decubitus position, the fluid is largely forced into the in-

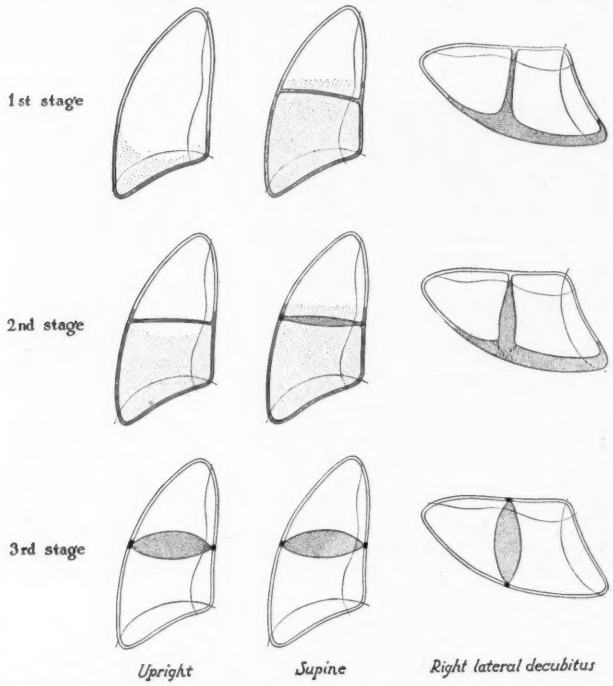

Fig. 7.—Mode of development of encapsulated interlobar effusion. (Minor fissure, right lung; major fissure omitted.)

Diagrammatic representation of findings in a large number of cases of pleural effusion, roentgenologically studied, to indicate the mechanism of formation of an encapsulated interlobar effusion. Note that, for purposes of clarity, only the minor interlobar fissure is shown. The pleura is represented by long, black lines, pleural space (exaggerated for purposes of illustration) by white, pleural fluid by stippling, and pleural adhesions by short, black lines.

The top row of figures indicates the roentgenologic appearance of a small effusion in different positions. With the patient upright, the fluid is largely below the dome of the diaphragm and does not reach up to the interlobar fissure. In the supine position, sufficient fluid extends upward to enter the fissure producing an horizontal linear shadow. In the lateral decubitus position, almost all the fluid is forced into the inferior costal gutter, reaching the minor interlobar fissure in considerable quantity. The fissure is separated, and the fluid enters into it producing a broader shadow.

The middle row is similar to the above except that fluid is present in larger amount. In the upright position there is sufficient to reach the fissure and produce a linear shadow. In the horizontal positions, the fissure shadow is much larger and begins to assume an oval shape in the lateral decubitus position. Pleural adhesions around the interlobar space may now begin to form.

In the bottom row, the final evolution of an encapsulated effusion is shown. The fluid from the general pleural cavity has been completely drained or absorbed while the interlobar space is completely closed off by adhesions. The shadow, therefore, is the same in all positions.

ferior costal gutter and a larger quantity comes in contact with the interlobar pleura, separating it to some extent and filling the space more fully.

During the second stage of an effusion there may be a sufficient quantity of fluid to reach the interlobar fissure even in the upright position, so that a fine linear shadow is produced. In the supine or prone position, the shadow becomes broader; while in the lateral decubitus position the shadow takes on the oval form of an encysted effusion. Meanwhile, in some cases adhesions begin to form. When these are complete, the interlobar pleural space becomes an entity, entirely independent of the general pleural space. At this third stage the absence of fluid in the free pleural cavity and the position of the patient do not appreciably affect the shadow. The visceral pleura lining this space may exude fluid and absorb it exactly as the peripheral pleura does. This is the final evolution of an encysted interlobar effusion. In the vast majority of instances spontaneous resorption occurs in the second stage and no encapsulation results. The development of adhesions is probably partly fortuitous, partly due to the character of the effusion.

With this hypothesis in mind, it can readily be seen that the presence of even a small amount of fluid in the free pleural cavity is a predisposing factor in the formation of encysted interlobar effusions. Furthermore, the position of the patient may be of some importance. The horizontal positions, particularly that one in which the patient lies on the affected side, would tend to favor this process.

SUMMARY

Roentgen observations on many cases of pleural effusion, on cases of pneumonia, and one detailed report of a patient with an encapsulated interlobar empyema are presented to demonstrate that:

1. Free pleural effusions may extend into the interlobar fissures particularly in the horizontal positions.
2. Roentgenologically these extensions may simulate encapsulations.
3. Encysted interlobar effusions may develop as a result of such an extension if adhesions form during the course of the effusion.
4. This may be the common mechanism for the development of the interlobar encapsulations which complicate pneumonia.

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DISCUSSION

DR. J. J. SINGER:—This excellent demonstration by Dr. Rigler shows more than ever the necessity of a closer cooperation between the surgical side and the medical and roentgenologic side of chest work . It is impossible for any one man to spend that much time on all phases of the subject to be able to see these beautiful details, which is the key to success in chest surgery.

The development of effusion in empyema and then catching these things at the right time will prevent so many of the cases that were described this morning under chronic empyema. We have recently at the Barnes Hospital in St. Louis had an illustration of careful study of encapsulated fluid. In this case it did not appear so much in the interlobar space, but elsewhere. It so happened that in the course of routine work, a patient was brought in for examination. After listening and fluoroscoping the patient, I made a diagnosis of fluid in the right chest. The physicians who brought the patient in looked at me with surprise and said, "Right chest? Why the fluid is in the left."

I said, "Well, you know there is always a difference of opinion. There is not very much, and I may be wrong, but I think it is in the right chest."

As happens in large institutions, it is always quite a thrill to have the consultant be wrong. The next day they brought the patient, with all glee, with fluid from the left chest. I saw the fluid; I saw where the needle was put in, and I put the patient in the fluoroscope and I said, "Yes, you got it out of the left chest, but you punctured the right pleural cavity through the left chest."

I put the patient on the right side, and the fluid was seen beautifully in the lateral side, and when the patient sat upright, the fluid disappeared, it did not enter the left lateral chest cavity as was shown. It proved to be that this fluid was in the vertebral gutter and in the upright position was almost entirely lost, but did so change the physical signs that there was dullness in the left chest due to the pushing over of the mediastinum into the left chest. So this demonstrates again the value of the close cooperation.

DR. CARLETON B. PEIRCE.—I should like to compliment Dr. Rigler on this excellent demonstration of a very simple procedure. Dr. Coryllos, I think, mentioned the "lordotic" position, which is extremely uncomfortable for the patient, and which is very awkward for the radiologist to use. The position which Dr. Rigler has suggested, however, and which he has used so nicely permits an examination of the patient with very little movement. He can be examined, if necessary, on his bed in his room in the hospital; whereas the other position requires a certain amount of special equipment and special handling of the patient.

I would suggest that this method be used more extensively. I thoroughly subscribe to what Dr. Rigler has shown in the development of these effusions which ordinarily are hidden in the usual routine examination.

DR. W. P. HERBERT.—I should like to ask Dr. Rigler a question. Dr. Rigler, do you feel in drawing out the fluid from that patient shown here that there was more possibility of encapsulation of the fluid in the interlobar space? It seems to me by drawing out the fluid you might get a sticking together of the interlobar space; whereas, if you had left the fluid in the pleural cavity, you would not have gotten such an encapsulation. I would like to have your opinion on this.

DR. LEO G. RIGLER.—I appreciate Dr. Singer's and Dr. Peirce's discussions very much. In regard to the question of withdrawal of fluid, I can realize the implications of that question, that is, that the withdrawal of fluid might cause the development of adhesions. I think it must be borne in mind, however, that with the needle we rarely if ever withdraw all the fluid which is present in the pleural cavity, and I hardly believe it would be possible to empty the pleural cavity so completely of fluid as to permit the formation of adhesions which otherwise would not have formed.

Our experience has been, for example, that even with closed drainage with a catheter we still find considerable amounts of fluid present in the pleural cavity, although suction has been used. I suppose, however, that the factor of withdrawal of fluid should be considered as a possible causative factor in the formation of adhesions.

OBSERVATIONS ON THE EFFECT OF HYPERVENTILATION ON THE VITAL CAPACITY OF SURGICAL PATIENTS

With Some Remarks on the Effect of Postoperative Complications and the Influence of Abdominal Binders and Adhesive Strapping*

John H. Powers, M.D.
Cooperstown, N. Y.

INCREASING interest in postoperative pulmonary complications during the past three decades is apparent in the more careful observation of surgical patients during this critical period, the greater frequency with which such complications are recognized, and the numerous therapeutic measures which have been suggested for their prevention and treatment. In an effort to approach the investigation of this subject from the standpoint of respiratory mechanics and the impairment thereto which follows surgical procedures within the abdomen, I carried out a series of pre- and postoperative observations of the vital capacity of surgical patients at the Lakeside Hospital in 1926.[1] Prior to the report of this work similar studies were published by Churchill and McNeil[2] and by Head.[3] Comparable results were obtained independently, and similar conclusions were drawn by all three investigators. A marked reduction in vital capacity was observed after all operations on the abdomen; the greatest reduction and most prolonged period of recovery followed upper abdominal incisions; a moderate drop and rapid recovery occurred after thyroidectomy; no significant changes were noted after other procedures in the neck, or after operations on the extremities and rectum. It was suggested that this postoperative respiratory impairment, the secondary pulmonary deflation and the associated reduction in vital capacity might play an important rôle in the unexplained relationship between postoperative pulmonary complications and abdominal surgery.

In addition to the patients who convalesced normally I had the opportunity to study a few individuals with postoperative complications, both pulmonary and intraabdominal. It was observed that pulmonary complications produce a secondary reduction in vital capacity by which their presence may be suspected before clinical symptoms appear and that inflammatory lesions and postoperative complications within the abdomen both diminish the vital capacity and prolong the period of recovery.

*From the Department of Surgery of the Mary Imogene Bassett Hospital.
Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery, New York, June 3, 4, 5, 1935.

Following the work of Henderson, Haggard, and Coburn[4] on the therapeutic use of carbon dioxide after anesthesia and operation, and of Henderson and Haggard[5] on hyperventilation of the lungs as a prophylactic measure for pneumonia, numerous surgical clinics adopted the routine use of carbon dioxide during the early postoperative convalescence in the hope of thereby diminishing the incidence of pulmonary complications. Successful results have been published by some authors and unfavorable ones by others. In a small series of patients, Sise and his collaborators[6] reported less decrease in diaphragmatic excursion in those treated postoperatively with carbon dioxide than in those who had no hyperventilation, but in the same paper he recorded a higher incidence of postoperative pulmonary complications in the treated than in the untreated group. So that on the whole the evidence for postoperative hyperventilation as a prophylactic measure against pulmonary complications is somewhat contradictory; theoretically the idea is attractive, but the results are unconvincing. King[7] carried out a precise and careful investigation at the Massachusetts General Hospital between December 1, 1930, and December 15, 1931. All the surgical patients after abdominal operations and hernial repairs were under his personal observation, and every other one was treated with carbon dioxide either through rebreathing tubes or administered by the Henderson inhalator. There was no significant difference in the total incidence of pulmonary complications in 648 patients so treated and in 667 control cases, and he concluded that inhalations of carbon dioxide were of no greater value in preventing postoperative pulmonary complications than frequent change in the position of the patient.

The question naturally arises, therefore, Is, or is not, the postoperative reduction in vital capacity following laparotomy influenced by hyperventilation?

Beecher[8] has reported observations from which he concluded that "carbon dioxide therapy following laparotomy has no effect in preventing the crippling of the respiratory system." The studies herein recorded have yielded contrary results and suggest a modification of this opinion.

METHOD

The Collins vital capacity spirometer was used. It consists of a delicately balanced bell floating in a cylinder of water. The most essential feature of the apparatus is the direct reading dial which is graduated to tenths of liters. Fractions of these gradations may be estimated in tenths also.

A nose clip was not used; the patient was instructed to close his nose by gentle pressure of the thumb and finger. Whenever leakage occurred, it could be easily detected by fogging on the polished metal surface of the mouthpiece.

All observations were made with the patient in a semi-sitting position and the head of the bed elevated to an angle of 45 degrees. They were also taken at least two hours after a meal in order to obviate the possible inhibitory effect of gastric distention.

The first observation was made before operation, and daily studies were continued throughout the convalescent period, beginning with the first postoperative day, until a high level was reached or the patient was discharged. The highest of three to five observations was chosen as the most accurate. A record was kept of the operative procedure, the type and the duration of the anesthesia.

Both male and female patients were studied. All were encouraged to exert themselves to the utmost in spite of pain, and I believe that the highest reading on any given day represents the patient's supreme inspiratory and expiratory achievement for that particular period in his or her convalescence.

No patients with drained wounds or postoperative complications, either above or below the diaphragm, were included in the first series of observations, which were made solely to determine the effect of hyperventilation on the postoperative vital capacity in uncomplicated cases. For that purpose the results have been separated into upper and lower abdominal groups and compared with those obtained in a comparable number of similar untreated cases studied in Cleveland.[1]

Hyperventilation was conducted by a trained anesthetist. A mixture of 5 per cent carbon dioxide and 95 per cent oxygen was delivered to the patient from a tank through a regulation tube and face mask. The first treatment was given at the termination of the anesthesia before the patient was removed from the table. Subsequent treatments were carried out on the ward three times daily for the first three postoperative days; each was continued until hyperpnea was produced, usually three to five minutes.

The postoperative complications, both pulmonary and abdominal, and their influence on the vital capacity, have been considered separately.

The studies to determine the effect of a many-tailed abdominal binder and adhesive strapping were made on patients who were in the hospital for study or because of infections or fractures of the extremities. Both the adhesive strapping and the binder were always applied by me in a manner similar to that which would be employed after an upper abdominal incision. The adhesive extended from below the umbilicus to the ensiform and the binder from the pubis to the level of the nipple. As with previous studies, the highest of five successive readings was chosen as the most accurate index of the vital capacity. The observations were made at least two hours after the previous meal and an interval of five to ten minutes for rest was allowed between each group.

RESULTS

1. *Effect of Hyperventilation.*

The uncomplicated cases in which carbon dioxide was used routinely during the first three postoperative days have been divided into two groups: upper abdominal in which incision was made through the right rectus muscle, and lower abdominal in which either a right rectus or a McBurney incision was used. All the data have been

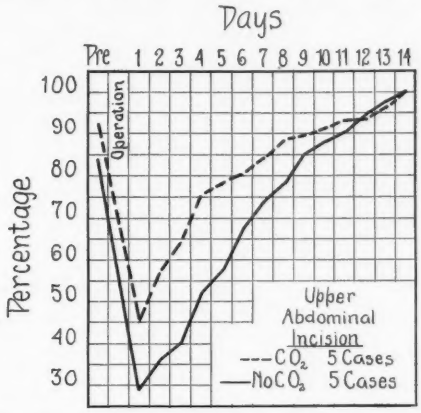

Fig. 1.—Graphic representation of the effect of pulmonary hyperventilation on the postoperative vital capacity of surgical patients after upper abdominal operations.

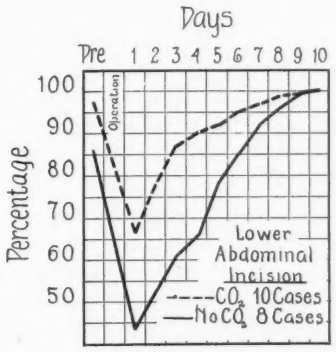

Fig. 2.—Graphic representation of the effect of hyperventilation on the postoperative vital capacity following lower abdominal incisions.

averaged and compared with similar groups of untreated patients who were studied in Cleveland several years ago.

In the upper abdominal series (Fig. 1) a difference of 16 to 23 per cent in the postoperative reduction in vital capacity occurred in the treated and untreated patients during the period of hyperventilation. (The lower preoperative average in the Lakeside group is due to the inclusion of one patient with a perforated duodenal ulcer.)

In the lower abdominal groups a difference of 23 to 26 per cent in favor of the treated series was found during the same postoperative interval (Fig. 2). The ultimate return to a high normal level, arbitrarily regarded as 100 per cent throughout these studies, was not hastened in either series, however, by the administration of carbon dioxide.

Fig. 3 is a graphic comparison of all the cases studied. The treated group includes 3 hernial incisions, 10 incisions in the right upper abdominal quadrant, 5 in the right lower quadrant, and 6 midline suprapubic incisions. In the untreated group are included 9 unilateral hernial repairs, 8 upper abdominal incisions, and 5 lower abdominal wounds. The average reduction in vital capacity during the first three postoperative days was 19 to 21 per cent less among the patients who received carbon dioxide.

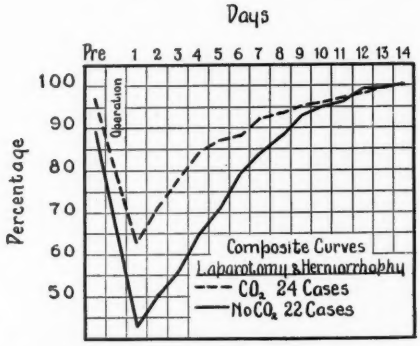

Fig. 3.—Composite curves determined by averaging all the observations for each day. Both the treated and the untreated groups include upper and lower abdominal, and inguinal incisions.

Comment. None of these series is large; comparisons have been drawn between data collected in two hospitals at fairly long distances from one another, one in a large metropolitan center and the other in a small rural community, yet the determinations have been made by the same individual, in the same manner, using the same kind of apparatus. The results suggest that less reduction in vital capacity occurs after abdominal operations if carbon dioxide is administered routinely during the first three days to induce pulmonary hyperventilation.

2. *Effect of Postoperative Complications.*

During the course of this investigation numerous patients developed postoperative complications of one sort or another, all of which produced alterations in the normal convalescent curve of the vital capacity. Some of these reflections were transitory, others prolonged.

Fig. 4 represents the effect of (a) chemical peritonitis following closure and plication of a perforated gastric ulcer, (b) postoperative hemorrhage into the intestinal tract after gastroenterostomy, and (c) bronchitis following cholecystectomy.

Fig. 5 discloses the unsuspected influence of a postoperative hematoma in a low right rectus wound on the third day, and the effect of transient nausea and vomiting, presumably due to temporary subacute intestinal obstruction, on the ninth day.

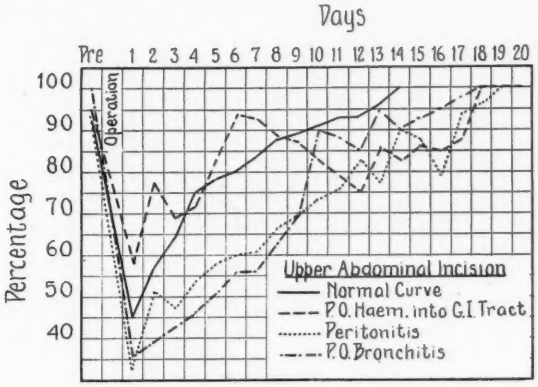

Fig. 4.—Graphic representation of the effect on the vital capacity of postoperative complications following upper abdominal surgery.

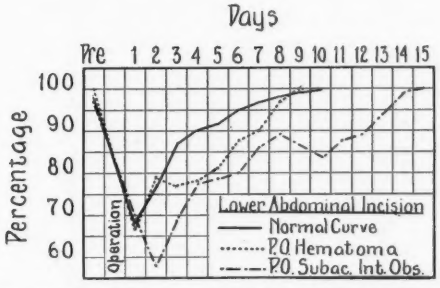

Fig. 5.—Graphic representation of the influence of abdominal complications after low right rectus incisions.

In Fig. 6 may be seen the very marked secondary reduction in vital capacity which occurs with postoperative massive atelectasis and the prolonged recovery following bronchopneumonia. Each of these complications was detected by the spirometer before clinical signs revealed the nature of the disease.

Comment. Accurate daily determinations of the vital capacity of surgical patients serve as a satisfactory criterion of their general postoperative condition.

3. *Influence of an Abdominal Binder, Adhesive Strapping, and Adhesive With Binder.*

In order to determine the effect on the vital capacity of a tight, abdominal, many-tailed binder, determinations were made before and after its application to patients who were admitted to the hospital for study, or because of infections or fractures of the extremities. The

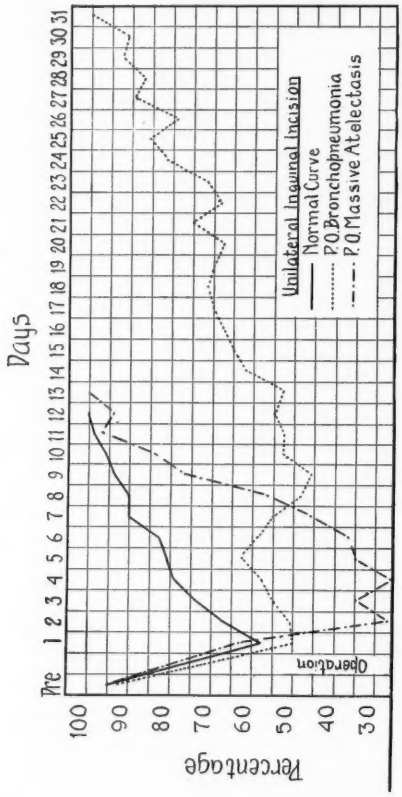
Unilateral Inguinal Incision
Normal Curve
P.O. Bronchopneumonia
P.O. Massive Atelectasis
Days
Pre 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
Operation
Percentage
100 90 80 70 60 50 40 30

Fig. 6.—Marked secondary reduction in vital capacity due to massive atelectasis of the lung, and prolonged recovery after bronchopneumonia following herniorrhaphy.

observations (Table I) suggest that such a binder plays a very small part in the postoperative reduction in vital capacity following laparotomy.

The influence of adhesive strapping simulating a constricting surgical dressing, and adhesive and binder in combination are presented in Table II. The results show no more restriction by adhesive strapping and binder together than by a binder alone.

Comment. These observations are not in accord with those of Churchill and McNeil[2] who reported a reduction of 30 per cent in

TABLE I

EFFECT OF ABDOMINAL BINDER ON VITAL CAPACITY

PATIENT	NORMAL VITAL CAPACITY	VITAL CAPACITY WITH BINDER	DIFFERENCE IN C.C.	DIFFERENCE IN PERCENTAGE
1	3854	3728	126	4
2	3470	3128	332	9
3	3310	3116	194	6
4	4332	4032	300	7
5	4740	4394	346	7
6	4612	3888	724	16
7	4366	4130	236	4
8	3774	3180	594	16
9	5176	4460	716	14
10	3734	3732	2	0
11	4536	4298	238	5
12	4584	4490	94	2
13	5300	5266	34	1
14	5322	5112	210	4
15	4282	4188	94	3
16	4540	4320	220	5
17	4806	4510	296	6
18	4070	3344	726	18
Average	4378	4074	304	7.1

TABLE II

EFFECT OF AN ABDOMINAL BINDER, ADHESIVE, AND ADHESIVE WITH BINDER

CASE	NORMAL	VITAL CAPACITY IN PERCENTAGE OF NORMAL		
		WITH BINDER	WITH ADHESIVE	WITH ADHESIVE AND BINDER
1	100	99	100	99
2	100	96	100	96
3	100	94	95	95
4	100	93	93	97
5	100	84	88	77
6	100	94	96	96
Average	100	93	95	93

the vital capacity of a normal individual by the application of a tight dressing over the right upper quadrant, held in place by an abdominal binder.

CONCLUSION

Less reduction in vital capacity occurs after abdominal operations if carbon dioxide is administered routinely during the first three days to induce pulmonary hyperventilation. Theoretically, this fact should have some bearing on the incidence of postoperative pulmonary complications if such complications be due in whole or in part to pulmonary atelectasis secondary to splinting of the abdominal musculature.

Postoperative thoracic and abdominal complications, even of mild severity, lower the vital capacity and prolong the period of recovery. Accurate daily determinations offer a satisfactory criterion of general postoperative convalescence.

Tight abdominal binders and adhesive strapping, singly or in combination, play very little part in the reduction of vital capacity after abdominal surgery.

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ASEPTIC ANEMIC INFARCT OF THE LUNG WITH SEQUESTRATION

Case Report*

I. A. Bigger, M.D., and George D. Vermilya, M.D.
Richmond, Va.

ANEMIC infarcts of the lung are of infrequent occurrence, as evidenced by the fact that Thomas[1] was able to find only fourteen such cases in the literature up to 1913; but Kaufman,[2] in his book on pathology, stated that he had seen several such cases. In all the cases collected by Thomas there was an associated suppuration, and we were unable to find a report of any case of aseptic anemic infarct of the lung.

In considering the mechanism of production of pulmonary infarcts, one must be familiar with the rather unusual anatomic arrangement of the pulmonary and bronchial arteries. The pulmonary arteries are not end-arteries; in fact, there is a rather free anastomosis between the branches of the pulmonary arteries especially in the deeper portions of the lung. There is no direct anastomosis between the bronchial and the pulmonary arteries, but there is an indirect communication between the bronchial circulation and the pulmonary circulation in that some of the bronchial veins, the so-called bronchopulmonary veins of LeFort, drain into pulmonary veins. Other bronchial veins empty into the azygi, intercostal veins, and the superior vena cava. This unusual anatomic arrangement of the vessels was described by Miller[3] and further substantiated by the work of Ghoreyeb and Karsner[4] who seemed to show conclusively by intravascular injections that when the pressure in the pulmonary circulation approached zero, blood flowed from the bronchial arteries through the capillaries into the adjacent pulmonary vessels. The anastomosis between the branches of the pulmonary arteries is much less free on the sharp edges and near the surface of the lung, and for this reason infarcts occur more frequently in these areas.

With these anatomic peculiarities in mind, one can readily understand why a simple occlusion of a branch of a pulmonary artery will not usually cause infarction in an otherwise healthy lung. If, however, the pulmonary circulation is already impaired, as for instance in pulmonary congestion secondary to insufficiency of the left side of the heart, an occlusion of a branch of a pulmonary artery may cause hemorrhagic infarction. This may also occur in the presence of an intense inflammatory reaction in the lung or in the presence of excessive fibrosis or emphysema interfering with the circulation in the capillaries.

*From the Department of Surgery of the Medical College of Virginia.
Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery, New York, June 3, 4, 5, 1935.

An anemic infarct can occur only as the result of a complete blockage of the circulation to a segment of the lung. It is quite natural to suppose that this would occur very infrequently, and it apparently does.

The case here presented is of especial interest, since it is an example of an aseptic anemic infarct of the lung occurring in an otherwise apparently healthy individual.

CASE REPORT

L. W., a colored man thirty-five years of age, was admitted to the Saint Philip Hospital on the medical service on December 15, 1932, complaining of a cough, hemoptysis, weakness, and loss of about twenty pounds in weight. He stated that until about one month before admission to the hospital he was in good general health. At that time he developed hoarseness, a relatively nonproductive cough, and pain in the left side of the chest. These symptoms persisted, and he became so weak that he was forced to give up his work about one week later. About a week after this, he began to cough up blood-streaked sputum. He stated that he had had an almost constant dizziness since the onset of the present illness.

There was an indefinite history of a luetic infection about twelve years before, and about ten years previous to the onset of the present illness he had received several intravenous injections of salvarsan.

Physical examination showed a well-developed, moderately well-nourished, colored man. Examination of the head and neck was negative except for rather severe pyorrhea and some injection of the pharyngeal mucous membrane. Eyegrounds were negative, ears negative. Inspection of the thorax showed some flattening and limitation of expansion of the left side. Percussion revealed dullness over the upper portion of the left lung, and auscultation revealed numerous bronchial and fine moist râles over the same area. Abdominal examination was negative; the extremities were negative; and there was no evidence of peripheral arteriosclerosis, varicose veins, or phlebitis. Neurologic examination was negative.

The blood pressure was 110 mm. Hg systolic, 80 mm. Hg diastolic; temperature 103° F.; pulse rate 120 per minute; respirations 25 per minute. Laboratory examination: Blood—red blood corpuscles 4,320,000; hemoglobin 80 per cent; white blood cells 6,600; polymorphonuclear leucocytes 70 per cent; lymphocytes 29 per cent; eosinophiles 1 per cent; Wassermann test repeatedly negative. Urine—negative save for a very slight trace of albumin. Sputum—negative for acid-fast bacilli on several examinations.

X-ray examination of the chest on December 16, 1932, the day following his admission, showed an area of consolidation in the upper portion

of the left lung, extending from the hilum to the periphery and between the first and the fourth ribs in front. A tentative diagnosis of pneumonia was made.

Course in the hospital: The patient's temperature fluctuated between 101° and 103° F. and his pulse rate remained elevated to around 120 per minute. The area of dullness slowly decreased in extent, and an-

Fig. 1. Fig. 2.

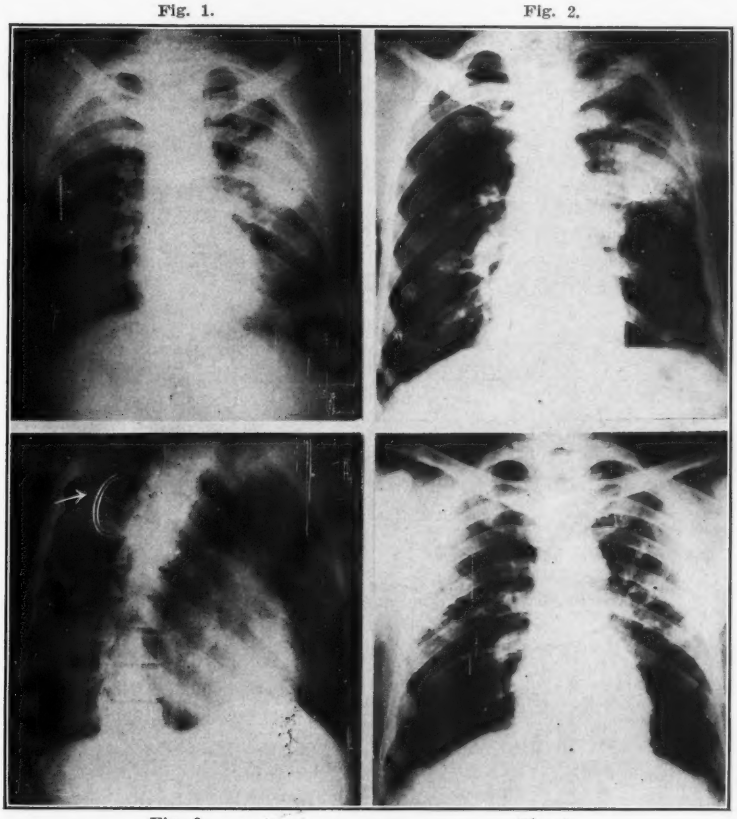

Fig. 3. Fig. 4.

Fig. 1.—Roentgenogram showing consolidation in the upper portion of the left lung at the time of admission to the hospital.

Fig. 2.—Roentgenogram of the chest made on January 26, showing the sequestrated infarct.

Fig. 3.—Roentgenogram of the chest made in the oblique position, showing the sequestrated infarct.

Fig. 4.—Roentgenogram of chest made on May 31, showing only a moderate amount of scar tissue in the left upper lung.

other x-ray examination on January 16, 1933, was reported as showing improvement in the appearance of the left lung, with a decrease both in size and in density of the shadow. Another x-ray examination on Janu-

ary 26 was reported as showing a dense, rounded shadow about 6 cm. in diameter in the upper portion of the left lung against the lateral chest

Fig. 5.

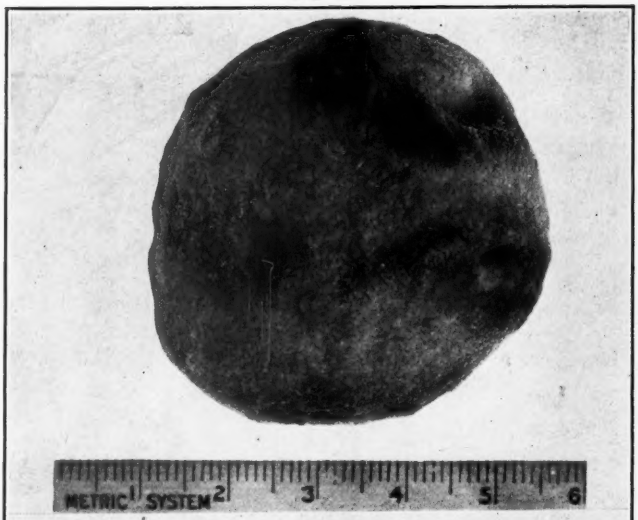

Fig. 6.

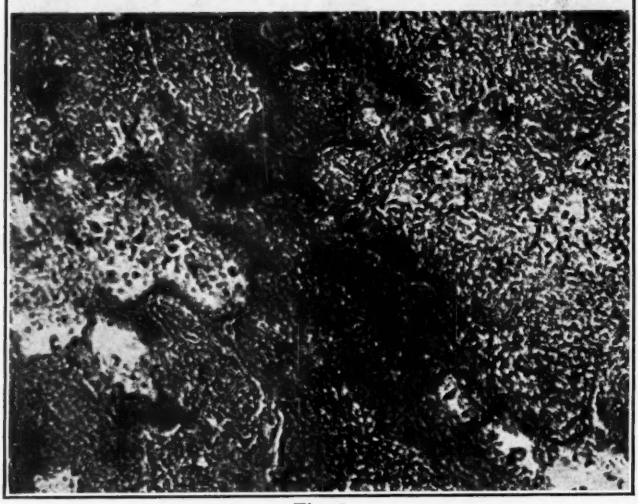

Fig. 5.—Photograph of infarct of lung.

Fig. 6.—Photomicrograph of the infarct, showing diffuse coagulative ischemic necrosis of pulmonary tissue in which the faint outline of alveoli can be seen.

wall. There was a dense outer ring surrounding a narrow clear zone and inside this a rounded area of increased density. The entire shadow was smaller than that on the previous examination.

On January 17 the patient complained of pain in the right ear, and examination of the drum showed that it was bulging. The left ear drum was normal. A right myringotomy was done on January 19 and a moderate amount of thin pus evacuated. X-ray examination of the right mastoid area on January 21 showed diffuse cloudiness over the mastoid cells but no evidence of bone destruction. The right ear drained freely for several days, after which the infection gradually cleared up.

On February 1 lateral and oblique films of the chest showed that the circular shadow was located about midway between the anterior and the posterior chest walls. At this time the patient's temperature was ranging between 100° and 101° F. and his leucocyte count between 11,000 and 14,000. A definite diagnosis could not be made, but several possibilities were considered, including benign tumor of the lung, echinococcus cyst, etc. Aspiration was done in the region of the shadow and a very small amount of thick fluid obtained which was examined for echinococcus hooklets but none could be found. At this time a surgical consultation was requested, and, although a definite diagnosis could not be made, exploration was advised because the shadow was so well circumscribed.

The patient was operated upon on February 8 under local infiltration anesthesia, one-half of 1 per cent novocaine solution. A perpendicular incision was made extending from the apex of the axilla down to the level of the lower border of the fourth rib in the midaxillary line. About 2.5 to 3 inches was resected from both the second and the third ribs; the intercostal structures were divided, and the parietal pleura exposed over an area about 2 inches in diameter, beneath which the lung was indurated. An incision was made through the pleura, both layers of which were thickened and densely adherent, and a smooth-walled cavity entered. It was almost filled by a yellowish gray, disk-shaped mass, which was attached by loose filmy adhesions. These were easily separated and the mass was removed. The cavity, which communicated with a bronchus, contained a very small amount of mucoid fluid. It was packed lightly with gauze and the incision in the chest wall partially closed.

The mass, which was 4.5 cm. in diameter, was of a rubbery consistency and had an irregular surface with markings characteristic of pulmonary tissue. The microscopic examination was reported by Dr. Lewis C. Pusch as follows: "Diffuse coagulative ischemic necrosis of pulmonary substance, in which the faint outlines of alveoli can be seen, without viable cells anywhere except in a narrow zone at the periphery, where a few leucocytes and collagenous fibers can be recognized." The tissue was stained for iron but none could be demonstrated. The diagnosis was aseptic anemic infarct of the lung.

Following operation the patient's condition showed little change, and his temperature fluctuated between 100° and 102° F. for about six

weeks. For ten days after operation the wound was kept lightly packed with gauze, which was changed at frequent intervals, but the drainage became more profuse, so a soft rubber tube was inserted into the cavity. X-ray examination on March 3 showed that the cavity had not decreased in size and contained a small amount of fluid, so it was decided that it would be wise to open the cavity widely and again pack it lightly with gauze. This was done on March 7, following which the patient's temperature rather promptly dropped to normal, and his subsequent course in the hospital was uneventful. At the time of his discharge on April 24, 1933, the cavity had been almost entirely obliterated, but a bronchial fistula was still present which did not completely heal until about three months later.

This patient was seen on March 1, 1935, and again on May 31, 1935, and found to be symptom free and apparently in excellent general health. Physical examination was negative save for the scar in the left axillary region.

DISCUSSION

The mechanism of production of the infarct in this case is difficult to explain. There was no evidence of cardiac disease or peripheral thrombophlebitis which might have given rise to emboli. The constant dizziness during the early stage of his illness and the fact that he developed a middle ear infection with mastoid involvement during his stay in the hospital suggest the lateral sinus as a possible source for emboli, but the absence of pain in the ear and the normal appearance of the ear drum at the time of admission seem to be strong evidence against such a possibility. The insidious onset suggests that it resulted from occlusion of some of the branches of the pulmonary artery by thrombosis. No cause for the development of thrombosis in the pulmonary vessels is apparent. The history of syphilis was not definite and the Wassermann test was repeatedly negative, so this was probably not a factor. Reports by Kessel,[5] Budinger,[6] and others indicate that thrombosis of the pulmonary vessels may complicate such conditions as pneumonia or foreign body in a bronchus, but no such predisposing conditions were demonstrated in this case, and suppuration, which would most likely have occurred in either of these conditions, was absent. This absence of suppuration is one of the most unusual and interesting features of this case.

SUMMARY

A case of aseptic anemic infarct of the lung with sequestration, occurring in an otherwise healthy individual, is reported. The sequestrum was removed surgically, and the patient subsequently recovered.

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DISCUSSION

DR. DEAN B. COLE.—I should like to show slides of two patients whom I have recently treated with artificial pneumothorax for "infarct of the lung."

(Slide 1.) White, male, farmer, aged thirty-one years, first seen in consultation Feb. 24, five days after appendectomy without drainage. The patient appeared to be acutely ill. Respiration rate of more than one hundred. Patient complained of severe pain in the right lower chest. He was treated symptomatically for five days when pneumothorax was instituted. Only two to three hundred cubic centimeters of air were given initially and no greater amount at subsequent refills. Patient's response was prompt, and he soon became symptom free. Pneumothorax has been continued and patient permitted to follow his occupation of farming. X-ray examination following onset of illness showed an atypical inflammatory lesion at the right base and indefinite haziness suggesting possible fluid. Present x-ray picture shows the lung practically clear.

(Slide 2.) White, male, merchant, aged twenty-five years. Seen May 13, 1935, with an atypical consolidation involving the lower half of the right lung. At this time the following history was obtained: Onset with measles in February, followed by pneumonia which was complicated by phlebitis of the right leg. Signs and symptoms of pneumonia disappeared and acute symptoms of phlebitis subsided after several weeks' rest. Patient had become ambulatory, returning to his business, when he became suddenly ill with acute pain in chest accompanied by marked dyspnea. This occurred one week prior to the time he was referred to us.

Patient was hospitalized and pneumothorax was instituted. He was relieved of his chest symptoms almost immediately, and was free of fever within two days, followed by a rise in fever three days later, which was accompanied by acute phlebitis. He has now returned to his home, is ambulatory and symptom free. Recent x-ray picture shows chest practically well.

Our experience confirms Dr. Brunn's in that the diagnosis of pulmonary embolus is not always easy and the choice of treatment is debatable. I feel that the diagnosis should be established before undertaking surgical intervention.

Dr. Ballon has raised the question as to the possible effect of pulmonic embolus on the central nervous system. A few months ago in consultation I saw a patient who was apparently dying of a pulmonary embolus following labor. She lived, but developed a severe empyema from which she also recovered. She soon developed a psychosis, which I thought was probably the result of anoxemia.

DR. I. A. BIGGER.—I wish to stress the fact that suppuration was not present either in or around this infarct.

Dr. Cole mentioned the use of pneumothorax in the treatment of patients with pulmonary infarction. Dr. Cole stated that he has given small amounts of air, sufficient only to separate the two pleural surfaces. I can see how this might give a great deal of relief from pain and believe that it should be a safe procedure. If the lung were actually collapsed, however, a portion of the clot might well be displaced and thus cause obstruction to other branches of the pulmonary artery.

MAGGOT AND ALLANTOIN THERAPY IN TUBERCULOUS AND NONTUBERCULOUS SUPPURATIVE LESIONS OF THE LUNG AND PLEURA*

Report of Eight Cases

Norman Bethune, M.B., F.R.C.S.(E.)
Montreal, Canada

SINCE Wm. S. Baer of Baltimore, in 1929, brought to the attention of the medical world the use of maggots of the blowfly in chronic osteomyelitis, with a paper entitled "A Viable Antiseptic,"[1] and followed this with a more important article in 1931,[2] there has appeared, mostly in the American literature, a number of articles bearing on this subject, the majority of which have concerned themselves with the treatment of chronic osteomyelitis. Scattered here and there are references to the employment of larvae in soft tissue infections also.

Goldstein[3] and Livingston[4] in 1932 both suggested they might be used in discharging wounds, leg ulcers, and inoperable carcinoma. Indeed, Smyth[5] in 1931 reported using maggots following electrocoagulation of cancer. Weil, Simon and Sweadner[6] used maggots in twenty-nine cases of soft tissue necrosis, including felons, tuberculous adenitis, inoperable cancer of breast, large areas of cellulites, infected abdominal incisions following appendix abscesses, infected amputation stumps with six cases of gangrene. All twenty-nine patients recovered, except one. They found it especially good in gas gangrene. Fine and Alexander[7] used maggots in three cases of diabetic carbuncle, two cases of traumatic injury, and two of decubitus ulcers with favorable results. They concluded that the maggots were particularly effective in soft tissue infections. Radabaugh[8] also reported their use in necrosis of the arm, and I have reported[9] a case of recurrent empyema treated by this method.

The following is a report of seven cases of pleural and pulmonary infections so treated, and one additional case treated with allantoin (the excretion of the maggot) as a control.

I. *Tuberculous empyema with bronchopleural, pleurocutaneous fistulas.*

Case 1.—E. L., No. 12491. Tuberculous pyopneumothorax following therapeutic pneumothorax. Repeated aspirations were followed by multiple infected needle tracts. Bronchopleural fistulas were demonstrated. Secondary infection—tubercle bacilli, staphylococci and diplococci. Costectomy and open drainage. Two applications

*From the Division of Thoracic Surgery and Bronchoscopy, Sacred Heart Hospital.
Read before the Eighteenth Annual Meeting of the American Association for Thoracic Surgery, New York, June 3, 4, 5, 1935.

of maggots were made through a large draining sinus. Quantitative count of tubercle bacilli in the pus fell from Gaffky VI to Gaffky II immediately. After two applications, started on daily irrigations of 0.5 per cent aqueous solution of allantoin. Gaffky rose again to VI. No complaint of discomfort. Patient did not cough up maggots. First maggots lived twelve days; second lot, only six days. Wound rapidly became alkaline. Patient gained two pounds in weight. Bronchopleural fistula still open. Hemogram showed increase of lymphocytes from 17 to 26 per cent. Total white cells (12,000) unchanged. Coagulation time of the blood fell from eight to six minutes after maggot therapy, and of blood when mixed with the discharge, from three to one and one-half minutes after maggots. Patient is permanently improved and now awaiting thoracoplasty.

CASE 2.—M. B., No. 17284. Spontaneous tuberculous pyopneumothorax with multiple (4) bronchopleural fistulas. Both sputum and pus contained tubercle bacilli and secondary invaders. Aspirated for three weeks. Closed three small fistulas by localized pleural poudrage with talc powder, under thoracoscopic control. One large fistula could not be closed by either powder or 35 per cent silver nitrate. Costectomy (2 ribs), and wide open drainage—size of opening 3 × 3 inches. Two applications of maggots at ten-day intervals, followed by daily irrigations with allantoin for one week. Implantation of maggots was followed by rise of temperature to 102° for forty-eight hours. Allantoin irrigations produced burning sensations in the wound. Discharge of pus, which was negligible after the maggots, increased following the allantoin irrigations and fell again when saline irrigations and intermittant applications of acriflavine and gentian violet were instituted. Hemogram showed rise of lymphocytes from 11 to 22 per cent. Total white cells were reduced slightly (20,000-18,000). Rapid disappearance of the tubercle bacilli, by direct smear, in the pus following the maggot treatment; but five weeks later, although smear was still negative, guinea pig inoculation was positive. Wound and pleura were very clean. Coagulation time of blood unchanged. Coagulation time of blood and pus before and after maggots—no change (two and one-half minutes)—but following allantoin irrigations, fell from two and one-half to one-half minutes.

Three months later, a total thoracoplasty (anterior and posterior), in four stages, completely obliterated the empyema cavity. Both pleurocutaneous and bronchopleural fistulas closed. Negative sputum. Patient recovered.

CASE 3.—J. C., No. 14039. Tuberculous pyopneumothorax following therapeutic pneumothorax. Bronchopleural fistula. Mixed infection. Tubercle bacilli and staphylococci in pus. Costectomy and open drainage. Total thoracoplasty (anterior and posterior) in four stages. Very thick pleura. Slitlike cavity remained. Two-stage Schedé opera-

tion. Partially open wound. Visceral pleura covered with slough. Six applications of maggots. After the fourth application, the maggots refused to stay in the wound, which had become remarkably clean. Coagulation of blood fell from nine to seven minutes. Coagulation of blood mixed with discharge before and after maggots fell from four to two minutes. Before maggot implantations pus was Gaffky II by direct smear. Tubercle bacilli could not be discovered by direct smear after maggot treatment, but six weeks later (after fifth application) guinea pig inoculation was still positive. Staphylococci remained unchanged in numbers. Following the maggots, allantoin irrigations were started. These did not clean the wound as well as saline or aqueous solution and dyes. Abandoned after one week. Hemogram progressively bad. Death in four months from reactivation of contralateral lesion, although the operation wound and pleura remained very clean.

II. *Tuberculous osteomyelitis of ribs with bronchocutaneous fistula.*

CASE 1.—O. F., No. 17077, aged fifty-five years. Cough and expectoration. Tubercle bacilli found in sputum. No obvious parenchymatous disease. Osteomyelitis of sixth and seventh ribs. Excised ribs proved tuberculous. Curetted bronchial fistula through periosteal bed. Left wound open. Discharge from wound contained tubercle bacilli. Three applications of maggots were given. Bronchial fistula did not reopen. Sputum negative for tubercle on smear. Wound became very clean after two applications of maggots. Allantoin irrigations after third application of maggots caused increase of discharge, and increase in Gaffky count. Maggots were never able to render discharge free from tubercle as seen by smear, but had reduced their numbers. Death in five months from disseminated disease.

III. *Tuberculous empyema treated by closed drainage and irrigations with allantoin.*

CASE 1.—L. D., No. 16262. Large tuberculous pyopneumothorax following spontaneous rupture of lung during pneumothorax. No bronchopleural fistula demonstrated. Pus contained only staphylococci on smear, although guinea pig inoculation was positive for tubercle bacilli. High fever. Closed drainage and daily irrigations given for two weeks with 0.5 per cent (saturated) aqueous solution of allantoin. No qualitative, quantitative, or bacteriologic change in the pleural exudate. Open drainage. Death from contralateral tuberculosis in three months.

IV. *Chronic nontuberculous recurrent empyema.*

CASE 1.—H. D., No. 16588, aged fifteen years. Previous empyema (three months) treated by costectomy and open drainage. Basic lesion is a lower lobe bronchiectasis. Wound healed in one month.

Empyema recurred. Wound reopened widely. Discharge very foul. Organisms: staphylococci, diplococci, and streptococci. No tubercle bacilli. One application of maggots. Wound became very clean and closed rapidly in fourteen days. Staphylococci were the last to disappear. No recurrence during six months' observation period. Coagulation time of blood fell from nine to seven minutes. When mixed with discharge from wound there was no change before and after maggots.

V. *Gangrene of lung.*

CASE 1.—E. C., No. 17478. Pulmonary gangrene involving lower half of left lung of five weeks' duration. Costectomy and cautery opening into central abscess, which was deep, and the size of an apple. Rapid gangrenous extension into chest wall. Bacteriology: three varieties of spirochetes, large and small fusiform bacilli, vibrio, diplococci and streptococci. Large amount of bloody, foul discharge. One implantation of maggots into wound. They refused to venture into the deeper recesses, but fed with avidity on the superficial aspects of the lung and chest wall. Within twenty-four hours they cleaned up this area remarkably, eating away the entire greenish gray slough covering the visible wound. Odor much less. The third day, on uncovering the dressing, the entire wound was filled with a stiff, solid blood clot. On removal of this clot, no visible bleeding point could be seen. It came from the depths of the lung cavity. Seven days later, patient died of sudden pulmonary hemorrhage by mouth, caused by ulceration of a branch of the pulmonary artery, close to hilus. No maggots were in the vicinity, but many were active on the superficial aspects of the wound, which was very clean. There was no evidence that the maggots were the cause of the terminal hemorrhage.

VI. *Abscess of the lung.*

CASE 1.—E. L., No. 17562. A single, circumscribed abscess cavity of right upper lobe, drained by the subapical branch of the eparterial bronchus. Collateral bronchiectasis. Posterior approach, costectomy and cautery pneumonotomy. Very foul discharge. One application of maggots into the cavity. Patient did not cough them up. The wound was deliberately kept open for two months to encourage lobar contraction. Healed completely. It is not thought that the maggots contributed a great deal to the successful result. They disliked the deep narrow sinus, and scampered out onto the surface when introduced. It is difficult to believe that they would reside with equanimity in the depths of a cavity.

COMMENTS

Seven additional cases to the one previously reported, of maggot therapy in suppurative lesions of the lung and pleura, and also one case treated with allantoin alone, are reported.

These comprise three cases of tuberculous empyema with bronchopleural, pleurocutaneous fistula: one cured following thoracoplasty, one permanently improved and awaiting thoracoplasty, and one dead of contralateral disease after showing temporary local improvement. One patient with tuberculous osteomyelitis with bronchocutaneous fistula showed temporary local improvement, and died of disseminated disease. One patient with recurrent nontuberculous empyema was cured. One patient with gangrene of the lung showed temporary improvement, and died of pulmonary hemorrhage unconnected with the specific therapy. One patient with abscess of the lung was cured. One patient with tuberculous empyema treated with allantoin irrigations was unimproved, and died.

Excluding the case treated by allantoin alone, and including the first reported case, a total of 8, the results are: cured, 4; permanently improved, 1; temporarily improved, followed by death, 3. In one of the cured, the specific therapy cannot be claimed to have played a more than minor rôle. In none of the deaths were the maggots, even remotely, causative agents.

Maggots will not venture into deep narrow sinus tracts; hence they are unsuitable for small openings into the lung or pleura. The whole wound must be wide open. They are surface feeders.

Bacterial laden pus is their natural food. They will eat only dead cells—the fouler the better. They do not cause hemorrhage (commented upon by Baron D. J. Larrey, quoted by Goldstein[12] in his observations on maggots in wounds during Napoleon's Egyptian campaign: "In fact, these larvae consumed putrid material and did not disturb any living tissue. No hemorrhages were ever observed under these circumstances, regardless of the depth to which the insects penetrated, or to the extent of the wound"). They will not live on healthy granulation tissue. Their presence is tolerated in wounds for only a comparatively short period of time. Possibly an antimaggot substance is produced in these wounds.

Maggots will ingest living tubercle bacilli, and their presence in the gut of the larvae is easily demonstrated. It would appear that the maggots simply suck them in with the free floating pus, or easily detachable dead cells. Some clinical evidence is presented which is suggestive that these bacilli are not simply excreted unchanged back into the wound. Otherwise, the number of bacilli as seen on direct smear of the pus should remain unchanged; whereas it was constantly reduced. The important point—the fate of the tubercle bacillus in the gut of the maggot—is under investigation at present, and no opinion can be ventured at this time.

Maggots are difficult to handle, and can be a great nuisance in chest cases. They are comparatively fragile organisms, difficult to breed unless great care and attention are given them and special apparatus

is used. They must be employed at just the proper period of their life cycle. They have a short life. No satisfactory cage was discovered to confine them in place. We used rubber, adhesive tape, liquid cement, collodion, Unna's paste, calamine lotion, Friar's balsam. All were more or less unsatisfactory.

A superior covering of the cage was devised, made from pieces of transparent, washed x-ray films, perforated for air, with a needle. This we found better than Baer's wire gauze netting, since being transparent a clear view of the wound is always obtainable.

In extrapulmonary, but intrathoracic, lesions, such as mixed infection tuberculous empyema with bronchopleural fistula, and nontuberculous foul empyema, they have given, in our hands, good results. They are a good preparation for subsequent thoracoplasty, as the comparatively clean pleuras resulting from their use will more readily adhere than those grossly infected.

In intrapulmonary lesions, such as gangrene or abscess, unless the cavity is laid wide open, their use is not advocated.

Robinson[11] discovered allantoin to be one of the natural excretions of the maggots and suggested its use in wounds. A saturated aqueous solution (0.5 per cent) seems to possess no bacteriocidal efficiency. It may be, as claimed first by Macalister,[10] a cell proliferant, but it will not kill tubercle bacilli, staphylococci, or streptococci. On the other hand, it seems harmless to the tissues.

Allantoin would appear to reduce the coagulation time of the blood. This may well be the reason why maggots have not been accused of causing hemorrhage in wounds in which they have been found, or placed. In four cases, we noted that the coagulation time of the patient's blood fell in three and remained unchanged in one, after maggot implantation.

Blood, when mixed with the wound discharges before and after maggots, fell in two, and remained unchanged in two. Blood, when mixed with pus, and after allantoin irrigations, fell in the one case it was checked.

In vitro, the same fall after allantoin was added to blood, was observed. Example:

Coagulation time of whole blood	7.0 min.
Coagulation time of blood and distilled water (5 c.c. plus 0.5 c.c.)	7.45 min.
Coagulation time of blood plus 0.5 per cent allantoin aqueous solution (5 c.c. plus 0.5 c.c.)	5.15 min.
Coagulation time of blood plus 0.5 per cent calc. chloride in distilled water	6.0 min.

Opposed to these observations is the experiment in which 10 grams of the crystalline allantoin was taken by mouth, on an empty stomach, and the coagulation time of the blood checked hourly for the next three hours. No change was observed. Also 10 c.c. of a 0.5 per cent

aqueous solution of allantoin injected intravenously did not affect the coagulation time of the experimenter's blood. Unfortunately, there was no opportunity of checking the urinary content of allantoin, following administration. Still, it is rather suggestive that the coagulation time of the blood of pregnant women falls at the same time as there is an increase of allantoin in their urine, which must be derived from an increased concentration of allantoin in the blood. This may, of course, be quite a fortuitous coincidence, and a result of other causes.

We have not had the opportunity of trying to stop a generalized oozing from a wound, by dusting onto the surface a finely ground-up powder of allantoin crystals, but it might possibly be found effective.

My sincerest thanks are given to Dr. Pare of St. Justine's Hospital, Montreal, for kindly supplying a large quantity of maggots; to the Mother Superior of the Sacred Heart Hospital for her financial support; to my assistants, and those nurses who were the harried guardians of this restless zoo; and last, but not least, to those patients who so willingly submitted themselves to this unpleasant form of treatment.

The first batch of maggots was supplied by Lederle Company of New York, and arrived through the mail in perfect condition.

The allantoin was supplied by the Eastman Kodak Company, Rochester, N. Y.

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DISCUSSION

DR. HARRY R. DECKER.—One would certainly feel from the presentation which Dr. Bethune has given us that there is certain indication for treatment of this sort. I do not feel qualified to speak with reference to the treatment as far as chest conditions are concerned, such as he has portrayed for us. I can only reason by analogy from work in osteomyelitis of the extremities. In our own work,

whereas we have found that the method is efficacious, in the average case it is disagreeable to the patient, and we are not able to train the maggots sufficiently well to stay in the wound. It is perfectly possible that Canadian maggots are more easily trainable and more easily hypnotized. I am very curious to know how many of them reach the upper respiratory tract through a fistula that may be there.

I think that treatment of this sort requires certain stolidity or unusual psychology on the part of the patient to tolerate it. Most of our patients have had sleepless nights when this treatment has been used, and have begged to have it stopped. As an alternative we have found that Dakinization will accomplish everything that maggot treatment will for necrotic osteomyelitis.

DR. NORMAN BETHUNE.—In reply to Dr. Decker about maggots reaching the upper respiratory tract, one of our patients with tuberculous empyema with multiple bronchopleural fistulas had three small fistulas within an area of a twenty-five-cent piece, and one very large fistula about the size of a pencil, but an inch away. We closed the three small fistulas, under thoracoscopic guidance, with talc powder. The big fistula did not close with the application of talc, nor did it close with the application of 35 per cent silver nitrate. We allowed ten days to pass before maggots were placed. The man coughed up two maggots in his sputum. He was quite amused by this. Really, I think objections to these maggots are purely psychologic ones. We are accustomed to thinking of the enemies of man as either small microorganisms, very little ones, and so dismissing them, or ones large enough, like the carnivorae which we can fight with gun or bow and arrow. Because maggots are in that intermediate stage, then they become objects merely of curiosity or dislike, but I believe it is an esthetic objection only from ages-old association with death and corruption. Incidentally, ten days after the coughing up of those maggots in the sputum, that bronchial fistula which had been open for a year closed. Why it closed, I do not know. I hope it did not close simply because it got blocked up with maggots!

Men do not mind it, except the occasional nervous patient. They do complain of tickling. We have tried to discover whether the tickling sensation is on the skin edge or whether it is actually in the wound. The maggot has a very tough skin. The body is segmented and along the ridges of these segments are little spikes. The maggot crawls by digging in with his spikes, so that a patient will occasionally complain of the tickling of these little animals at work. They also complain, however, of the burning sensation of the allantoin, because we put in the allantoin after we had used all the maggots.

Incidentally, they were not Canadian-born maggots; they were American maggots naturalized.

After the maggots are used, the wound becomes comparatively clean. We had some delay in maggot production, and used allantoin. We have tried to tempt the maggots back into the wound by the utilization of their own excreta; that is, allantoin. The last man on the moving picture film in which the maggots were flying out of the wound in all directions had been on the allantoin irrigation, but they did not like even that. I think there is possibly some antimaggot production going on in the wound.

We are now thinking that if we can find a bactericidal agent that will kill the tubercle bacilli and put it in combination with cell-stimulating allantoin, we shall have a happy combination. Whether that is possible or not, we do not know.

As to the question of whether or not the maggots kill the bacilli, we have not found any positive experimental evidence as yet.

The Compressionist's Creed

I believe in Trudeau, the mighty father of the American Sanatorium, maker of a heaven on earth for the tuberculous; and in Artificial Pneumothorax; which was conceived by Carson; born of the labors of Forlanini; suffered under Pompous Pride and Prejudice; was criticized by the Cranks whose patients are dead and buried; thousands now well, even in the third stage, rose again from their bed; ascending into the Heaven of Medicine's Immortals, they sit on the right hand of Hippocrates our Father, from thence they do judge those phthisiotherapists quick to collapse cavities or dead on their job.

I believe in Rodington, Brehmer, Koch and Brauer, in Murphy, Friedrich, Wilms, Sauerbruch, Stuertz and Jacobeus, in the unforgiveness of the sins of omission in Collapse Therapy, in the resurrection of a healthy body from a diseased one and long life for the tuberculous with care everlasting.

Amen.

The editor prevailed upon Dr. Norman Bethune, the author of The Compressionist's Creed, to allow its publication in the Journal.

Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

Two Cases of Early Carcinoma of the Larynx, and a Number of Cases of So-Called Adenoma of the Bronchi Apparently Cured by Dia-Thermy. John D. Kernan. Laryngoscope **45**: 760, 1935.

The author presents ten most instructive cases. The chief symptom of a patient with a benign adenoma of a bronchus is apparently hemorrhage; other symptoms and the physical signs are dependent upon and consequent to acute or gradual, partial or complete bronchial obstruction. They therefore vary accordingly. The roentgen ray appearance of these tumors is variable; in one case there was a globular mass which had "very little effect upon the appearance of the rest of the lung." In another case the appearance was that of atelectasis. The bronchoscopic appearance of these tumors is "just as characteristic of these tumors as is their microscopic appearance and their place of origin." They are soft looking, movable and have "a suggestion of a pedicle about them. They are inclined to be vascular and bleed rather easily on contact with the bronchoscopic tube." Apparently only very exceptionally do they cause fixation of a bronchus.

These tumors grow with a broad pedicle from the bronchial mucous membrane into the lumen of the bronchus. Only late do they have a tendency to break through the bronchial wall and invade the lung tissue. These tumors never metastasize to other organs. It is worthy of note that Kernan previously reported a case of benign tumor of a bronchus as a carcinoma cured by diathermy and implantation of radon seeds.

The microscopic picture of one benign adenoma of a bronchus is said to be characteristic of them all. It is one of anastomosing columns of cells which in places have a tendency to form glandlike structures. A more detailed description is not furnished in this presentation.

Treatment: Disregarding the consequences of long-standing bronchial obstruction Kernan's results were as follows: In one instance the

tumor disappeared spontaneously; one patient was relieved of her symptoms—but not of the tumor—following x-ray therapy. Eight patients were cured by diathermy; two of these had radon seeds implanted as well. Two of the patients died following lobectomy for bronchiectasis. In one of these latter cases the diathermy contributed to the persistence of the bronchostenosis.

Harry C. Ballon.

Diaphragmatic Abnormalities. T. Wood Clarke, et al. Arch. Ped. **52**: 798, 1935.

Diaphragmatic abnormalities are divided into five categories: (1) true hernia, congenital in type, in which there is a developmental defect in the diaphragm and the abdominal organs protrude through the muscular layer and are contained in a hernial sac; (2) next in frequency is the false diaphragmatic hernia which is either congenital or traumatic, and in which there is no sac present and the protruded abdominal organs are in immediate contact with the lungs and pericardium; (3) eventration of the diaphragm, in which this organ, due to paralysis of one phrenic nerve or the congenital absence or atrophy of the muscles of one-half of the diaphragm, is extremely thin and weak and is arched high in the chest so that the stomach and intestines while below the diaphragm are in close proximity to the heart and lungs; (4) congenital absence of one-half of the diaphragm, a condition rarely recognized (only fourteen cases reported in the literature); (5) a condition known as "thoracic stomach" which is most rare, wherein the esophagus being very short, the stomach is placed above the diaphragm, which is intact, and the pylorus or duodenum passes into the abdomen through the esophageal opening in the diaphragm; only six of these cases are reported in the literature.

The authors report a case of absence of the diaphragm and a case of eventration of the diaphragm. These conditions are often misdiagnosed as diaphragmatic hernia, and the authors warn against the intervention of surgery in these cases. These two conditions, they report, are also sometimes misdiagnosed as hydropneumothorax, and if a needle is inserted peritonits may occur.

Henry L. Cabitt.

Pulmonary Syphilis in Adults, With Report of a Case. W. W. Robinson. Radiology **25**: Nov., 1935.

The author reviews the literature concerning luetic lesions in the lung fields. The clinical criteria upon which a diagnosis of pulmonary syphilis may be made are: (1) the patient must have a luetic history; (2) there must be signs and symptoms of a chronic, progressive pulmonary lesion; (3) the sputum must be negative for tubercle bacilli on

repeated examinations; (4) a positive serology; (5) a definite lesion by x-ray examination showing a response to antiluetic treatment.

There is no typical x-ray picture of pulmonary syphilis. The author describes three types as seen by x-ray: (1) a generalized thickening of all bronchial markings studded with minute "knobby" enlargements radiating out from the hilus in one or both lung fields producing a characteristic fan-shaped effect; (2) gumma—miliary, multiple or solitary; (3) diffuse lobar form.

The author reports an undoubted case of pumonary syphilis involving the upper and lower lobes of the left lung. In this case all the clinical criteria were confirmed, and there was a prompt and lasting response to specific therapy.

The possibility of confusing these cases with pulmonary carcinoma or tuberculosis is emphasized.

Henry L. Cabitt.

Sternoclavicular Branchioma. B. N. Fried. Am. J. Cancer **25**: 738, 1935.

The author describes two cases showing a unilateral neuritis of the brachial plexus, a homolateral Horner's syndrome and an atrophic monoplegia of the corresponding arm.

Roentgen examination of the affected side showed a dense shadow confined to the region of the first three ribs.

Postmortem examination revealed a squamous epithelial cancer originating in the region of the left sternoclavicular articulation, invading the infraclavicular and supraclavicular fossae, clavicle and upper three ribs. The pleura, lungs and other viscera were free from tumor.

The author divides the clinical aspects into: (1) the "silent" stage; (2) the stage of transitory or fugitive reactive neuritis; (3) the stage of outward swelling accompanied by a constant neuritis of the brachial plexus with a Horner's syndrome. The symptomatology, however, is not specific, since these syndromes may occur in cases having metastatic lesions in this area, carcinoma of the thymus or bronchogenic carcinoma. What is significant is the combination of a dense apical shadow with homolateral brachial plexus neuritis. One should then suspect primary carcinoma of the lung or extrapulmonary carcinoma of the type described in this article.

Pathologic study indicates that the tumor probably is of a low grade malignancy so that it is imperative to trace the disease at its onset for the advisability of surgical intervention or the use of x-ray therapy.

The probable origin of these tumors is from epithelial rests of the lower clefts of the branchial apparatus. These tumors are therefore named sternoclavicular branchiomas.

Henry L. Cabitt.

Acute Empyema in Children. J. M. Mason. J. A. M. A. **105**: 1114, 1935.

This is a report of 103 patients with empyema seen between January 1, 1929, and June 1, 1935. One hundred and two of these patients were treated surgically with mortality of 7.76 per cent. Since the war, the lowered mortality rate is due more to better understanding of treatment than to the presence of less virulent organisms. Any proposed methods of treatment of empyema should meet three requirements: reduction in mortality, a shorter period of convalescence, and a reasonable certainty of cure. Cases treated by aspiration alone, aspiration and replacement with air, aspiration followed by instillations of ethylhydrocupreine, and those treated by closed drainage alone do not fulfill these requirements as well as does open drainage with rib resection. It is best to perform repeated aspirations until a true abscess has formed; at this time a rib resection should be done in the most dependent portion of the abscess. Local anesthesia is preferred. The tube inserted at operation should be of large caliber; as the cavity decreases in size, smaller tubes should be inserted. The progress of the case should be followed by frequent x-ray films and fluoroscopic examinations. Any rise in temperature indicates a too rapid removal of the tube with a collection of pus, an encapsulated pocket of pus elsewhere, or some other complications as otitis media or a gastrointestinal upset. The treatment of empyema in children is no different from that followed in adults.

W. D. Thompson, Jr.

Fibrin Bodies in Artificial Pneumothorax. Henry K. Taylor, and I. D. Bobrowitz. Radiology **25**: 274, 1935.

This is a report of fibrin bodies observed in 94 patients, seen during the past two years, who received collapse therapy by artificial pneumothorax for a malignant type of pulmonary tuberculosis. All but three of these cases had a pleural effusion. Of the 91 cases with pleural effusion, the fibrin bodies appeared subsequent to the development of the effusion in 74; the effusion and the fibrin bodies were observed simultaneously in 15. In the remaining 2, the fibrin bodies preceded the pleural effusion by several months. These fibrin bodies are usually found in the lower portion of the pleural space. They may be free or attached to the chest wall, the lung, the pericardium, or the diaphragm. Not infrequently they have multiple attachments. Most of them though attached do have partial mobility. In only four cases were multiple bodies found. These fibrin bodies cast dense shadows and assume various shapes; however, irrespective of their shape, the majority of them have smooth outlines. They vary also in size, the longest diameter being 8 cm. They occasionally disappear, but the

nature of their disappearance is unknown. Fibrin bodies were removed from six patients while thoracoscopic examinations were being performed. The microscopic study of the sections made from four of the removed bodies presented a central nucleus of dense compact fibrin arranged in concentric layers. Where the fibrin was most dense, there was an absence of intact cells. The cells enmeshed between the fibrin at the periphery of the fibrin body apparently were derived from blood. The presence of the fibroblasts and histiocytes at the periphery of the fibrin body was explained by an attempt at organization of the mass. It was concluded that the fibrin bodies arose from the blood elements. From the complete analysis of the cases it was assumed that these bodies developed from fibrin derived from (1) blood, (2) pleural effusion, or (3) both.

W. D. Thompson, Jr.

The American Association for Thoracic Surgery

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Nineteenth Annual Meeting of the American Association for Thoracic Surgery

The next meeting of the American Association for Thoracic Surgery will be held at the Mayo Clinic, Rochester, Minn., on May 4, 5, and 6. The hotel headquarters will be The Kahler. It is suggested that members of the Association and visitors correspond directly with the manager of the hotel for reservations. The annual dinner will be held on Tuesday evening, May 5, at The Kahler. The list of members on the Local Committee for the meeting follows: Stuart Harrington, Chairman; W. S. Lemon, Herman Moersch, Porter Vinson, Howard Gray, and Harry Wood.

The Program Committee wishes to emphasize at this meeting the discussion of Nontuberculous Lesions of the Chest. Although papers dealing with any aspect of thoracic surgery will be welcome, those having to do with the esophagus, intrathoracic tumors, and the surgery of the heart and lungs are particularly desired.

In order to arrange the progam effectively and to facilitate the discussion of papers at the meeting, members are requested to send short abstracts as well as titles of their papers to the secretary before March 1. With this cooperation it will be possible to send our comprehensive programs in April and thus allow time for the preparation of discussions.

The final program will be published in the April issue of the JOURNAL OF THORACIC SURGERY.

Arrangements will be made by the Local Committee for the entertainment of the wives of visiting members and guests. Visitors who are interested in thoracic surgery are welcome to attend the meeting.